OCT 1 1919

Vol. I.—No. 3. MARCH, 1917.

THE BRITISH JOURNAL OF OPHTHALMOLOGY,

INCORPORATING

THE ROYAL LONDON OPHTHALMIC HOSPITAL REPORTS,

THE OPHTHALMIC REVIEW,

AND

THE OPHTHALMOSCOPE.

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LONDON:
PRINTED AND PUBLISHED FOR THE BRITISH JOURNAL OF OPHTHALMOLOGY, LTD.,
BY GEO. PULMAN & SONS, LTD., AT 24, Thayer Street, Marylebone, W.

Issued Monthly: Single Copy 4 shillings net:
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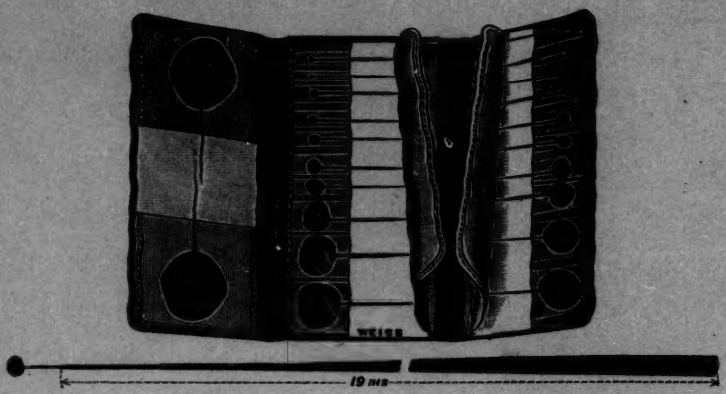

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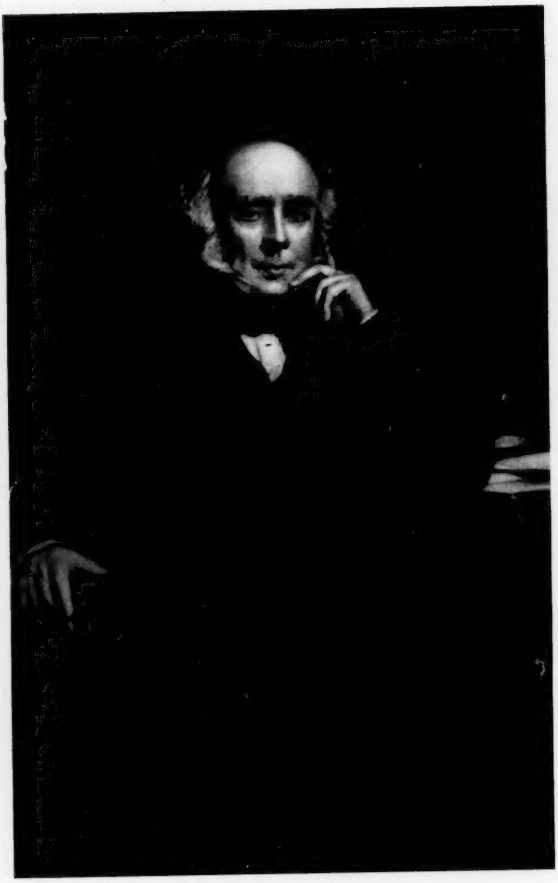

WILLIAM McKENZIE.
(1791—1868)

Whilst going to press, we learn with profound sorrow of the death, after a brief illness, of WALTER HAMILTON HYLTON JESSOP, President of the Ophthalmological Society of the United Kingdom, and Managing Director of The British Journal of Ophthalmology. We shall publish an account of his life and work in our next number.

THE BRITISH JOURNAL
OF
OPHTHALMOLOGY.
MARCH, 1917.

COMMUNICATIONS.

BRITISH MASTERS OF OPHTHALMOLOGY SERIES.

I.—Sketch of the LIFE OF WILLIAM McKENZIE, M.D. (1791-1868.)

Her Majesty's Oculist for Scotland.
Surgeon to the Glasgow Eye Infirmary.

BY

FREELAND FERGUS, M.D., F.R.S. Ed.

GLASGOW.

GLASGOW with the West of Scotland is, in the popular mind, chiefly associated with industrial development, the rule of Bailies, and bad weather, but its contributions to the intellectual developments of the country have been not inconsiderable. Thus, in engineering science it has produced men like James Watt, McQuhorn Rankin, James Thomson, and Lord Kelvin. In philosophy it has had Thomas Reid, the founder of the Scottish school of metaphysics, Adam Smith, the political economist, Edward and John Caird, and many others of considerable note. In medicine, it has been served by a race of men remarkable for clearness of vision and originality of thought. True it is that many eminent Glasgow and Lanarkshire men have sought fame in regions far removed from the places of their upbringing and many of them have risen to great distinction, even in the Metropolis of the Empire. The birth places of William and John Hunter, of Cullen, of Matthew Baillie and of David Livingstone are all situated within a distance of three miles from each other and in close proximity to Glasgow. Cullen was born at

Hamilton, his father being the chamberlain to the then Duke of Hamilton. Matthew Baillie was the son of the Minister of Bothwell, and the brother of Joanna Baillie, the poetess. The Hunters were the sons of a small landed proprietor in the parish of East Kilbride, and David Livingstone was the son of a weaver in the village of Blantyre.

Amongst other names associated with the Glasgow School of Medicine we may mention Joseph Black, Thomas Thomson, Thomas Graham, afterwards Master of the Mint, and Joseph Lister. It was during his occupancy of the Chair of Surgery in Glasgow that Joseph Lister really put his finger on the diseased spot and said that wounds went wrong because of micro-organic life. No greater discovery has ever been made in the annals of humanity, and it gives the discoverer a foremost place in the ranks of those that have benefited the human race. It was while he lived in Glasgow that Lord Lister evolved the theory that failure from inflammation after operation depended on micro-organisms and was therefore preventable. In the early sixties perhaps no School in the country was better equipped than Glasgow. Allen Thomson taught anatomy; Sir William Gairdner, practice of medicine; Joseph Lister, surgery; John Easton (of syrup fame), materia medica; and William McKenzie, ophthalmology.

William McKenzie was all his lifetime, except for a short period when he was abroad and in London, connected with the City of Glasgow. His father was a manufacturer and a Glasgow merchant, and in the same city his distinguished son was born in the year 1791. He received his education in the Grammar School of his native place and in its University. When he entered Glasgow University, it was with a view of studying for the Presbyterian Ministry of the Church of Scotland, and, as a matter of fact, he had gone the length of enrolling as a student in the theological classes of that ancient seat of learning. What changed the course of events cannot now be ascertained.

His son and his widow are long since dead, as are also, so far as the present writer knows, his most intimate friends, such as George Rainy and the late James Robertson. He had not gone far in his divinity studies until he gave them up altogether and entered the medical classes. So far as can be learned, his medical studies were chiefly carried out at the University of Glasgow and at the Glasgow Royal Infirmary, where, as an undergraduate he was a pupil of Dr. Richard Millar, who subsequently was the first professor of materia medica in the University, and was, in addition, a man of considerable and wide erudition, for he made several contributions to the study of the history of medicine which are even yet appreciated as valuable. There is reason to believe that McKenzie began his medical curriculum about the year 1810 and qualified to practise by

taking the diploma of the Faculty of Physicians and Surgeons of Glasgow, in the year 1815. In the days of which we speak the profession was seldom entered, even in Scotland, by means of the Universities. Few were the University medical graduates to be found in practice, even in the larger towns of Scotland. Most men entered the profession by means of an apprenticeship and by the licence or diploma of such bodies as the Royal Colleges in Edinburgh and the Faculty of Physicians and Surgeons in Glasgow. Shortly after taking the diploma already mentioned, McKenzie determined to go further afield, and from 1815 to 1817 or 1818, he spent his time in London and on the Continent, chiefly in Paris, Pavia, and Vienna.

While on the Continent, he must have studied very widely, for he attended Roux in Paris, and Beer in Vienna, and also studied medical jurisprudence under Orfila. He was back in London probably about the end of 1817, or early in 1818, and settled in the Metropolis of the British Empire with the view of practising ophthalmology. He became a member of the Royal College of Surgeons of England. His best biographer, George Rainy, to whose monograph the present writer is indebted for most of the facts concerning the life of McKenzie, indicates that he was disappointed in his candidature for a popular lectureship on the subject of anatomy, and that this led to his returning to his native city in the year 1819.

Throughout his life, McKenzie was a diligent student. He read everything connected with medicine and with his own speciality on which he could lay his hand and carefully made notes of all the vast amount of literature which he perused. Moreover, he was an extensive writer, and thus we find that so early as his residence in London he made a contribution on "Diseases of the Lachrymal Organs." So far as the present writer is aware, that was his sole contribution to medical literature during his London career.

On his return to Glasgow he at once started as a general practitioner, with, be it said, a strong bias to ophthalmic practice, for within five years of his return to his native city, he, in company with Dr. Monteath, started in 1824, in a very small way, the Glasgow Eye Infirmary. He was appointed to the Chair of Surgery and also to that of Anatomy in Anderson's College, Glasgow, immediately on his coming to the city.

He always had a great interest in anatomical research, and to the last believed that a sound knowledge of anatomy was the essential basis of medical knowledge. In these days there was no Anatomy Act and the supply of material was extremely deficient, so we find McKenzie in 1824 issuing "An Appeal to the Public and to the Legislature on the necessity of affording dead bodies to the Schools of Anatomy by Legislative Enactment." This was published

in Glasgow in 1824. In this *brochure* he gave a detailed scheme almost precisely similar to the one that was subsequently included in Warburton's Anatomy Act. He held the Chairs of Anatomy and Surgery in Anderson's College until the year 1828. In that year he was appointed lecturer in the University of Glasgow on the "Structure, Functions, and Diseases of the Eye," which lectureship he held till his death in 1868. During his tenure of the office of the Professorship of Anatomy he produced a monograph on Human Myology.

In addition to lecturing on surgery and anatomy, he also gave systematic teaching on materia medica and medical jurisprudence, and, further, gave a course of lectures on the ear.

McKenzie, as already mentioned, was a prolific contributor to medical literature, but the work which brought his name prominently before the then ophthalmic world was unquestionably his "Practical Treatise on the Diseases of the Eye." That first appeared in 1830. It was translated twice into French, and into German, and Rainy also states that it was translated into Italian, although there seems to be some doubt as to whether the Italian translation was ever printed. It ran through four English editions, the first appearing, as already said, in 1830 and the last in 1854.

McKenzie also published a book on the "Physiology of Vision." This appeared in 1841 and is well worth perusing, were it only for the fact that it is a serious attempt to found a knowledge of this special subject on a physical basis. The earlier chapters of the work contain a very fair description of physical optics, as then understood. Is it too much to hope that at some time or other the modern student of ophthalmology will base his study of the eye on the same sure foundation? The list of authors at the end of each chapter of this work shows how thoroughly McKenzie had at his finger ends the bibliography of ophthalmology. Thus we come across the names of the men who were giants at that time, and before it, such as Thomas Young, Baptista Porta, Poggendorf, David Brewster, and a host of others. McKenzie certainly had a thoroughly reliable knowledge of the literature of his subject, and evidently was well-acquainted with its scientific side, as then understood.

In the current literature of the time we find many of his contributions. He was editor, for a period, of the *Glasgow Medical Journal*, to which he largely contributed, and also sent contributions to many other medical and scientific journals. Thus, in 1845, he made a contribution in the *Edinburgh Medical and Surgical Journal* on the "Vision of Objects on and in the Eye," which was an introduction to the then little known science of catoptrics.

We venture to think that McKenzie has left practically nothing which will be permanently associated with his name. He was

unquestionably a man of great learning, not merely in the science of medicine but in other subjects. He had an excellent knowledge both of the classical and modern languages and was in all respects a widely-read and well-informed man. At the same time it must be freely admitted, he was not an investigator. He was more a collector and a brilliant clinician for the time at which he lived than a researcher. He was a shrewd observer and took the greatest pains to write down carefully all the clinical facts of his cases both in his private practice and in his public, but we venture to say that experimental research was entirely alien to his bent of mind. In an earlier part of this communication it was mentioned that at one period of his life he gave lectures on medical jurisprudence, but as he found himself unable to manipulate the experimental parts of the course, he handed over that portion of his subject to a colleague. But McKenzie's book, although entirely out of date, cannot fail to be interesting to anyone who studies the history of ophthalmology. It was perhaps the last of the great books published before the scientific period began. A careful perusal of its pages will at any rate be useful to the modern practitioner, for it will show him the entire change that has come over medicine in all its branches within the last fifty years. Two discoveries about the middle of last century absolutely changed ophthalmic practice. One was the introduction into practical use of the ophthalmoscope by Helmholtz, about the year 1851, and the other was the discovery by Joseph Lister, at that time a colleague with McKenzie in Glasgow, some time before the year 1869, that post-operative inflammation was of micro-organic origin.

In those days the success or failure of a cataract operation was supposed to depend upon a number of circumstances, to which no operator now-a-days pays the slightest heed. The season of the year was one of them and the careful previous preparation of the patient for days or weeks beforehand was another. It was not realized that apart from occurrences which no man can foresee, such as intra-ocular hæmorrhage, the prognosis of a cataract operation very largely depends upon the flora of the conjunctival sac. It is interesting to find that so recently as 1854 McKenzie still selected as probably the best operation for cataract that of displacement, which he described under two categories, *viz.*, depression and reclination. In the main, he was not favourable to extraction. He evidently thought it highly dangerous, on account of its liability to be followed by a suppurative inflammation.

So everywhere it is the old-world teaching. Ophthalmia tarsi was to be treated with bread and milk poultices. The chief remedies to be employed under almost all circumstances were bleeding, mercury, and setons. He lived in the age of what was called the anti-phlogistic treatment and he carried it out with

vigour and determination. In his later years, however, he to some extent abandoned this practice. It is almost pathetic to find in the description of his cases in his great work how he would draw so many ounces of blood and yet make, a day or two afterwards, a note on the same case that, notwithstanding the remedies, the patient was no better. Had he been a young man when Joseph Lister taught, it probably would have occurred to him that he was working entirely on wrong lines. Ophthalmology had not become then as it has now, a practical application of three sciences: physics, physiology, and pathology.

McKenzie, according to the light of the time, still believed that it was of vital importance what salts were dissolved in the fluid that was to be used for an eye wash. There was no germ theory in his time, and, therefore, no practitioner of McKenzie's day ever for a moment supposed that the only important constituent of any lotion is water which, if not to be painful, must be made isotonic with something or other. Ointments and lotions and emplastra formed his chief armamentarium. Every now and again in his book the careful reader stumbles upon a case which was treated with leeches and with various other things of that sort where probably the sufferer was only the subject of some form of undetected refraction error or muscular irregularity. Yet there is no doubt that McKenzie was well acquainted with the physical side of the subject, as then understood. In the fourth edition of his book he gives no less than four pages to the subject of astigmatism, in which part of his work he details the two well-known cases of Thomas Young and Airy, the Astronomer Royal. The treatment of eye diseases was still supposed to be, for the most part, a matter of pharmacopœal remedies properly applied. Helmholtz at that time was living and working, so also was Donders, but the mass of ophthalmic practitioners throughout the world had not realised how completely the old order was to pass away in but a few years. McKenzie, however, was certainly well abreast of the times in which he lived.

Perhaps two things may be attributed to him. He was, we think, probably one of the first to give definite and special information on sympathetic ophthalmitis, and, in the second place, he very early in his career gave a definite description of glaucoma. We do not claim for him priority in the description of sympathetic ophthalmitis. Other writers had previously discussed the matter, but certainly McKenzie's description of the symptomatology of sympathetic ophthalmitis leaves nothing to be desired. At the time of the publication of his book, enucleation as a preventive measure was not in existence, although even then it was known that if the eye primarily affected were early destroyed, sympathetic ophthalmitis did not take place in the second eye. The destruction

of the originally wounded eye was often brought about by incisions and the application of poultices and such like means, and McKenzie advocates that where an eye is so severely injured as to involve risk to the other, that this line of treatment should be adopted.

In dealing with glaucoma, his description of the symptomatology is almost classical, and he was amongst the earliest to draw attention to increase of tension as being a characteristic symptom of the disease. He thought the chief cause of glaucoma lay in a diseased condition of the lens.

It only remains to be added that McKenzie became an M.D. of the University of Glasgow in the year 1833, and was admitted a Fellow of the Royal College of Surgeons of England, ***honoris causa***, in the year 1843. Modern ophthalmology in any true sense of that term he never knew, but he certainly was a brilliant exponent of the science as it existed in his time, and unquestionably his book on the physiology of vision shows that his bent was very largely scientific. By one thing his name is still kept alive in Glasgow and the West of Scotland, and that is by the lotion which he used universally. It was practically a solution of sal alembroth, sometimes made up with a little belladonna. These two lotions are still extensively used by some of the surgeons of the Glasgow Eye Infirmary, under the names respectively of "simple" or "compound lotion," and even yet in chemists' shops in Glasgow prescriptions are often received simply for McKenzie's lotion.

It may also be not inappropriate to mention that McKenzie's usual fee for a consultation was 3s. 6d., for which sum the patient was not only seen by McKenzie and advised, but also received a bottle of one or other of the foregoing lotions from an attendant who kept his consulting rooms.

A man who knew him well told the present writer that on one occasion a gentleman had travelled half the world over to consult McKenzie. At the end of the interview he put down a gold guinea-piece on the consulting room table, whereupon the great man immediately dipped his hand into his pocket, got out a lot of change, and counted out 16s. 6d. Such were fees in those days charged by the Royal oculist in Scotland, and yet, at his death, McKenzie left a very considerable fortune.

He is said to have been a man of friendly manners, possessed of a very refreshing, homely, mother-wit. As a young boy, the present writer, being a school companion of McKenzie's son, remembers him quite well, but was at that time much too young to remember anything personally about him except that he appeared to be a very genial and kindly man to youngsters.

ON THE RULES FOR THE CORRECTION OF ASTIGMATISM.

BY

J. W. NORDENSON,

LECTURER ON OPHTHALMOLOGY AT THE ROYAL CAROLINE INSTITUTE, STOCKHOLM.

ALTHOUGH the question, whether in a given case the astigmatism of an eye should be corrected or not, occurs daily, and is important and complex, it has been little debated in ophthalmic literature, and the text-books give only vague directions on the subject. I venture, therefore, to review shortly the rules for and against the correction of astigmatism.

Indications for the correction of astigmatism.

In general, correction is needed only if it increases visual acuity. An exception to this rule will be mentioned later when the contra-indications are being discussed. When correction improves acuity, it may be advisable on account of the following:—

I. *The need of accurate detail-vision.*—This point is so obvious that it hardly needs discussion. It should be remembered, however, that, although a visual acuity considerably below normal is sufficient for most occupations, the education of the human mind is based largely on visual perceptions, and the more accurate these are the better. It is, therefore, often right to give correction, although the vision without it is sufficient for the patient's occupation. This applies, of course, more to children than to older persons.

II. *Asthenopia*—chiefly of two kinds:—

(*a*) *Accommodation asthenopia.*—In accommodating the pupil contracts; this cuts off the peripheral rays, and thereby tends to improve definition. But some ametropes, in order to obtain the maximum of this effect, accommodate in excess of what gives the proper focal distance. This spoils definition, and so counteracts the good effect of the contraction of the pupil. The patient, therefore, relaxes accommodation. The pupil thereby widening, he reverts to accommodation. If this alternation goes on for long, it produces fatigue and strain—accommodation asthenopia.

(*b*) *Eyelid asthenopia.*—The second kind of asthenopia is due to the strain of the orbicularis muscle. This muscle can be used to correct astigmatism, its palpebral portion exerting pressure on the globe. As this pressure cannot be exerted in any but a vertical direction, it is only inverse astigmatism that can be thus corrected. In this way as much as 2 or 3 dioptres can be compensated, but the continual straining of the orbicularis leads to fatigue, and may constitute an urgent need for cylindrical glasses.

Asthenopia arising from astigmatism has been considered a common cause of such nervous troubles as epilepsy, migraine,

headache, etc., and it cannot be denied that there is a connection between these affections and astigmatism, and this is a good reason for correction in these cases.

III. *Dangers to the eye involved by astigmatism.*—Astigmatism may favour the development of strabismus or of myopia.

The development of strabismus is often due to the fact that the faculty of fusion is strained at the time when the desire for single vision is diminished. This diminishing of the desire for single vision occurs if one eye has less sharp retinal images than the other. The patient then suppresses the images of the defective eye, and takes no trouble to direct it so as to obtain single vision; he leaves it to take the position most convenient to its muscles.

Astigmatism in one eye, or astigmatism of different degrees in the two eyes, may, therefore, cause strabismus. It is, therefore, important that astigmatism should be corrected as soon as strabismus seems imminent. In certain cases the correction may at the same time help to prevent amblyopia ex anopsia. And if strabismus has already developed, the correction is likewise indicated as tending to promote fusion.

As to the relations between astigmatism and myopia, opinions depend ultimately on the views held as to the ætiology of myopia. It is generally admitted that close work plays a prominent part in the development of myopia. Astigmatism, by lowering visual acuity, induces the patient to bring the work close to the eyes, or the eyes down to the work; so, if myopia shows signs of developing, the astigmatism should be corrected. And in the case of inverse astigmatism, if the patient uses the orbicularis in the way described above, this, too, may promote myopia.

Contra-indications to correction.

I. *The inconvenience of wearing glasses.*—Only the case in which one eye is normal and the other astigmatic need be discussed here. In such cases it is generally not necessary to correct; sometimes, however, it is necessary, as, for instance, if the astigmatism is causing asthenopia, or if strabismus is threatening. Further, it might be of use to correct the single astigmatic eye if the patient needs very accurate binocular vision or a very wide field of vision, as might be the case with military men, for instance. If for some other reason the question of correction of the other eye occurs, astigmatism in the one eye further indicates correction.

II. *Non-improvement of visual acuity by correction.*—If an eye, though astigmatic, sees no better when corrected, it is generally useless to prescribe the cylinder unless it is required for some other purpose. And, if useless, glasses are an unnecessary expense and trouble. Sometimes, however, it may be advisable to correct even such an eye, namely, an eye in which correction has apparently no

effect on acuity. One often meets with cases of astigmatism, particularly of direct astigmatism, in which correction gives at first no improvement. If, in these cases, correcting glasses be worn notwithstanding, one finds after a time, sometimes months, sometimes years, the patient comes back with considerably better acuity; it may be increased, for instance, from 6/24 to 6/8 or better. This fact has been observed by many colleagues, but as far as I have seen, it has been very little discussed in the literature.

How is this increase of visual acuity to be accounted for? First, all sources of error must be excluded. There are mainly two such sources: (1) Very often at the first examination a patient unacquainted with the methods does not give exact answers, and does not read the test types, which he is quite well able to distinguish. It is well known to every practitioner, that especially children, when examined again on a subsequent day, often show decidedly better acuity. In the cases of this kind which I have seen, and from which I have attempted to draw any conclusion of this sort, the examination has, therefore, always been repeated at least once some days later. (2) The other source of error is variation in the size of the pupil. If the pupil is of different width on the two occasions on which the patient is examined, this will influence the degree of astigmatism found, and the acuity of vision. It must, therefore, be ascertained that the pupil is of the same diameter on both occasions. I have done this by Haab's method, which seems sufficiently accurate for this purpose.

These sources of error having been excluded, it seems justifiable to suppose that the acuity has really been increased. The explanation seems to be as follows:—When the eye is corrected the retinal image becomes sharper and the diffusion-circles smaller. Now a person who has been long accustomed to large diffusion-circles has probaby developed a faculty of interpreting them. When these diffusion-circles are suddenly reduced he will not at first be able to interpret fully the sharper retinal image. But after some time, when he is familiar with the new conditions, he will read more of the test-types.

The fact that correction of astigmatism does not immediately improve acuity is not, therefore, a final contra-indication to correction, which latter should be omitted only if the acuity remains unimproved after the use of glasses for some time. It seems advisable, therefore, especially in young children, to try correction for some time, and discontinue it if it proves useless.

III. *Intolerance of correction.*—In many cases the patient cannot tolerate correction, although this has been accurately determined and gives a considerable increase of acuity. This fact is often observed. Burnett* writes as follows: "It is a fact,

*Burnett, S. W.—"A Treatise on Astigmatism," p. 163. St. Louis, 1887.

however, which occurs with unfortunate frequency in our clinical experience, that glasses which give perfect optical correction, particularly if it is determined under atropin, cannot be worn with comfort and sometimes not at all. Such cases are to be found as a rule in persons who have passed their youth with their anomaly uncorrected." Burnett endeavours to explain them. "I can only account for them," he says (p. 164), "on the supposition that the eyes have been accustomed for so long to the astigmatic state as to make the abnormal, in a certain sense, the normal condition, and that the cerebral centre for vision resents an interference with the established order of things. It must be remembered in this connection that in dealing with the human eye we have to do not with an optical instrument alone, but with an organ of sense as well. All of our senses are in a measure affected by education, and after a certain habit has once been firmly fixed it is with difficulty changed in any important particulars."

These opinions are illustrated by two cases out of his own practice, and probably all oculists could confirm them from their own experience. It occurs fairly often that a patient cannot tolerate the correction prescribed for his astigmatic eye, and the explanation given above seems to be a very likely one. It occurs mostly with patients of a certain age, and especially in those cases where the axes of the optical system of the eye do not coincide with the vertical and horizontal plane through the system, but run in a more or less diagonal direction. These patients are accustomed to distorted images in which right-angles appear acute or obtuse, and vertical and horizontal lines appear more or less diagonal; but they know by experience the real shape of the objects thus misrepresented; their cerebral centre of vision has learnt to interpret the images correctly. In such a case when the astigmatism is first corrected the correct retinal image is misinterpreted, so that a right-angle appears acute or obtuse.

But the mechanism of interpretation can be re-modelled to a certain extent, and the patient succeeds in a short time in interpreting correctly the new retinal image. But in some people this is only attained after considerable trouble. As is the case with all faculties depending on exercise, it is far easier for the young person than for an elderly one to re-educate his mechanism of interpretation. It is therefore only natural that the difficulties should increase with age.

The question is now whether such patients should be encouraged to try to accustom themselves to the correction or whether it should be discontinued. The age of the patient and the nature of his occupation will decide. Elderly people should generally not be pressed to continue to wear the correction in such cases, as they often seem to suffer a great deal from it. In young people one should persuade them to persevere, as it often happens that after

some weeks, they have accustomed themselves to the glasses. In these cases it may often be advisable to correct the astigmatism by degrees, giving a weak cylinder first, letting the patient accustom himself to it, and gradually increasing it. In giving glasses for presbyopia the correction of a monocular astigmatism is generally not tolerated by the patient, and should be omitted. The same applies often to the correction of young hypermetropes for close work.

IV.—In a remarkable article Lagrange* pleads very urgently for not correcting astigmatism in children. He cites a number of cases in which astigmatism had been found and glasses prescribed but not worn, and in which, after a lapse of time, the patients returned with better vision and less corneal astigmatism than before. He is of opinion that this self-cure of astigmatism is due to the generally admitted tendency of the eye to become inversely astigmatic in old age, and as he has found no such change of astigmatism in eyes that had been properly corrected he considers correction an impediment to this natural alteration of the eye. The mechanism of this spontaneous cure of astigmatism consists, according to him, in the unequal contraction of the ciliary muscle in different meridians, this muscle pulling most strongly in the horizontal meridian, and thus increasing the curvature of the cornea in this meridian. He, therefore, advises not to correct the hypermetropic astigmatism so often found in children unless it is causing definite trouble. If it is causing trouble, spherical glasses should be ordered first, so as to oblige the patient still to make efforts of astigmatic accommodation. If, however, the spherical correction should prove insufficient, one should proceed sparingly to cylindrical correction, and be satisfied as soon as the troubles of the patient are relieved, and his acuity of vision is sufficient for his work.

In the light of our modern conceptions of the mechanism of accommodation, this explanation of the self-cure of astigmatism seems untenable. The theory appears to be based on too few cases. For the present the possibility of self-cure by leaving the astigmatism uncorrected is not sufficient to contra-indicate correction in these cases.

The above indications and contra-indications do not exhaust the list; moreover, they are, of course, not absolute, but must be weighed one against another according to the individual needs of every case.

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*Lagrange.—*Archives d'Ophtalmologie*, 1909, p. 705.

QUININE AMAUROSIS WITH REPORT OF A CASE.*

BY

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OPHTHALMOLOGICAL literature contains reports of a great many cases of quinine amblyopia or amaurosis, yet the condition must be of relatively rare occurrence in this country. The writer has seen three cases in the course of fourteen years, but this is certainly an unusually large number for such a short experience. Uhthoff[1] tells us that he has met with only one case among 100,000 patients.

The history and clinical aspect of the "typical" case of quinine blindness are familiar to all. The patient, in a fit of carelessness or recklessness, swallows a quantity of quinine sulphate, usually much in excess of the pharmacopœial dose. Within a few hours there is tinnitus, deafness, and bilateral blindness. Occasionally there is collapse, delirium, convulsions, or loss of consciousness. Very soon the cerebral symptoms pass off, then the hearing returns, and the patient is left with a visual disturbance very often amounting to total blindness, and with widely dilated and unresponsive pupils. In almost all cases, when the first ophthalmoscopic examination is made, the discs are found to be pale and all the vessels contracted, the arteries often reduced to almost invisible threads. Sometimes the central part of the fundus has a pale cloudy aspect, with a cherry-red spot representing the fovea. After an interval, varying from a few hours up to several weeks, light perception and the pupil reactions begin to return. At first the light perception is only transient, and this feature characterises also the form vision, the return of which soon follows. The recovery of function is at first purely central. In course of time the field of vision expands, but although central vision in many, if not most, cases becomes normal in a few weeks, there remains a permanent concentric contraction of the field. There is also found to be a degree of night-blindness, which, added to the contraction of the field, hampers the patient in spite of the excellent visual acuity. Of the retinal changes, the retinal opacity and foveal spot disappear and the vessels may regain their calibre to some extent, but narrowness of the vessels and a pronounced pallor of the discs are permanent.

The facts of the case now to be reported differed sufficiently from this description to warrant some comment:

The patient, a girl 22 years of age, stated that she had been feeling "run down," and had been taking quinine in the form of pills and powder for some weeks, the powder being in a packet from which she helped herself to a small dose from time to time. On

*Read at a Meeting of the Oxford Ophthalmological Congress, 14th July, 1916.

25th November, 1915, on account of a bad attack of toothache, she took a large dose of the powder. She estimated this at two teaspoonfuls, but there is some doubt as to the accuracy of her statement. In a few hours she felt stupid, with noises in her ears, deafness, and blindness. Her doctor, who was called in, found her in a semi-conscious state, which he attributed to an hysterical fit. He was told the quinine story, but did not think it very reliable. She was admitted to the Glasgow Eye Infirmary, on account of the continued blindness, on 29th November; that is, four days after the onset of her symptoms. I found her mental condition quite clear and her hearing normal, and there was no complaint of tinnitus. Both pupils were widely dilated and quite inactive. Blindness was absolute. She could not see an electric light held close to the eyes, although she could feel its heat. The fundi were practically normal. There was, perhaps, a little haziness of the nasal border of each disc, and some streakiness of the retina along the course of the vessels leaving the temporal side of the discs.

Next day the conditions were unaltered. Further examination of the pupils showed that the orbicularis reflex was present, but neither light, near vision, nor sensory reflexes could be elicited. The girl was of a slightly pasty, anæmic complexion, but well-nourished and apparently placid in temperament. The pulse was 66, temperature normal, and systolic blood pressure 80 mm. Hg. There was a slight cyanosis of the hands. No sensory or motor disturbance existed anywhere, and the heart was normal. The ophthalmoscopic examination was repeated every other day, and on 4th December, nine days after the ingestion of the quinine, and five days after the first examination, the discs were found to be pale and all the vessels abnormally pale and narrow. There was still no vision, and the pupil light-reaction was very doubtful, even with strong focal illumination. Twenty-four hours later, vision began to return. During the brightest time of the day she recognized the face of a nurse and other light objects, but in the dull light of the afternoon she could see nothing. Next day she could count fingers with each eye at one metre, but the field of vision was strictly central and very small, and the function of the retina seemed to be readily exhausted. There was steady improvement in vision from day to day, and a gradual expansion of the field, but at the same time an increasing pallor of the discs and narrowing of the vessels. A week after admission the pulse was 72 and systolic blood pressure 100 mm. Hg.

She was dismissed at the end of December, and had then practically reached the condition in which she is now, seven months from the onset of her symptoms. There have been slight fluctuations the V.A., probably owing to variations in illumination at the times

of testing, her best being, with each eye, 4/9, Snellen, and No. 1, Jaeger. The right pupil is slightly smaller than the left. The reactions are now fairly active. The field of vision shows marked concentric contraction, its maximum extent being 40 degrees from the fixation point in the temporal direction. Central colour vision is normal. She experiences considerable difficulty in finding her way about in the dark. The discs are very pale, like ivory with a delicate suggestion of a pink colour. Both arteries and veins are narrower than normally, the arteries, especially where they cross the discs, being pale in colour.

Comments.

It will be noticed that this case differs from the great majority of those hitherto published, in that, although the patient was first seen four days after the administration of the quinine, a further period of five days elapsed before the characteristic ischæmia of the disc and retinal vessels made its appearance, and it is this feature of the case to which I desire to direct attention, since it seems to have some bearing on the pathogenesis of the condition. An observation of this kind is not unique, but it is very exceptional. Although the statement is occasionally met with in text-books and elsewhere that quinine amaurosis may occur with a normal condition of the fundi, the cases on which the statement is based are not easy to find. The first two cases, one of them unilateral, were reported by v. Graefe[2], and the conditions in these cases were so unusual that Uhthoff[1] doubts their being due to quinine. Browne[3] mentions a case in which contraction of the vessels was first seen on the eighth day, although an examination had been made as early as the fourth day. Again, there is a case seen by Muntedam[4] in which, five days after the ingestion of the poison, there was œdema of the retina, the disc was not pale, the vessels were not distinctly narrowed, but there was absolute blindness and the pupils were widely dilated. In this instance pallor of the disc was first seen on the twenty-second day, light perception began to return on the twenty-sixth day, and, as vision recovered, the discs grew paler and paler and the arteries became very narrow.

I have not been able to make an exhaustive search of the literature, but so far as it is accessible to me at present these are the only references I have found to quinine amaurosis with a normal, or almost normal, condition of the fundi. In the vast majority of published cases it is clear from the details given that, from the date of the first ophthalmoscopic examination onwards, pallor of the discs and narrowing of the arteries were regularly present.

It is possible that the infrequency with which negative findings have been reported is to some extent due to the fact that the first ophthalmoscopic examination is not made sufficiently early; indeed,

in looking through the published reports one is surprised to find how seldom an examination has been made within a few hours, or even within a few days, of the onset of the blindness. But it would not be safe to assume that in every case a period elapses between the occurrence of the amaurosis and the onset of fundus changes, for there are records of observations even earlier than in my own case, for instance, those of Grüning[5], Parker[6], Behse[7], and Manolescu[8], in all of which fundus changes were already present as early as from 12 to 48 hours after the poisoning. Another possibility suggests itself, namely, that some of the objective appearances described belong to what might be called the acute stage, others to the later or chronic stage, and that there is usually a gradual transition from one to the other, but that in some few cases the two groups of changes are separated by an interval during which conditions are apparently normal. There is something to be said for this view. For example, when a cloudy pallor and cherry-red spot have been seen to occupy the central part of the fundus, they have been early phenomena, and they have always passed off —usually within a few days. There are other cases in which the earlier conditions were suggestive of retinal hyperæmia, and the later changes were in the direction of ischæmia. Then, from the experimental side, there is the statement of Druault[9], that we can distinguish between a primary anæmia, which comes on (in dogs) in six to seven hours, and disappears in twenty-four hours, and a secondary anæmia which appears on the fourth or fifth day, increases up to the twentieth or thirtieth day, and is permanent. It is likely that earlier and more systematic examination of the fundi would show the occurrence of a negative period to be less exceptional than it has hitherto appeared to be, but this has been so seldom observed that the current view of the pathogenesis of quinine amaurosis practically ignores it, and is based on the assumption that ischæmia of the vessels of the optic nerve and retina is the earliest result of the ingestion of quinine, that it is the cause of the amaurosis, and that it persists, the pallor of the nerve passing through time into a condition of true atrophy. This view is supposed to be confirmed on the pathological side by the experimental work of de Schweinitz[11, 12], Holden[13], and others, but neither the clinical nor the histological foundations of the theory are complete.

Turning first to the clinical aspect, it will be admitted that even a single case of the kind here reported would be enough to prove that the presence of ischæmia is not essential for the amaurosis; but even if one considers only the "typical" cases, one must be struck with the want of parallelism between the ischæmic phenomena and the state of the vision. It may almost be said to be the rule for the pallor of the disc and the attenuation of the retinal vessels to

become more and more pronounced during the very period when vision is returning. The other fundus changes sometimes described, such as blurring of the disc margins, congestion of the retinal vessels, œdema of the retina, the red foveal spot, etc., even if they were always present, are transient, and it is still less easy to establish a correspondence between them and the profound loss of vision which characterises the early stages of the affection.

For our knowledge of the pathological histology of quinine amaurosis, we are almost entirely dependent on the results of animal experiments, for example, those of Druault, Ward Holden, and de Schweinitz, in which dogs were employed. All are pretty well agreed that at a comparatively early stage degenerative changes can be observed in the ganglion cells of the retina, while the later changes comprise an ascending atrophy of the nerve fibres traceable as far up as the external geniculate body, and organic changes in the retinal vessels in the form of endovasculitis, etc. Holden's[13] view of the pathogenesis of the condition is that there is first a pronounced ischæmia of the optic nerve and retina, that this leads to degeneration of the retinal ganglion cells and nerve fibres (the immediate cause of the amaurosis), and that this, in turn, leads to ascending atrophy of the optic nerve. Druault[9,10], on the other hand, says that the degenerative changes are due to the direct toxic action of the quinine on the ganglion cells, and that the retinal ischæmia has little or no significance for the amaurosis. We have already seen that he distinguishes between a primary anæmia of the papilla and retina, which is transient, and a secondary, permanent, anæmia, which, in his view, is the result, and not the cause, of the cytological changes. Holden's theory, or something like it, is the one most generally accepted, but is unsatisfactory in so far as it fails to explain the few cases in which ophthalmoscopic examination has given a negative result. In such a case as the one I have described above, one may say with confidence that, if ischæmia of the retina and nerve is the first link in the pathogenic chain, it must have been such a transient ischæmia as Druault refers to. And no one can readily believe that a temporary interference with the nutrition of the retina might bring about changes in the ganglion cells which would persist, even after the circulation was restored. I am not aware that Druault's observation has been confirmed in any human case of quinine poisoning. A single case observed at the earliest possible moment, and kept under careful observation until the final state was reached, might clear up the whole question. On the other hand, if such a transient ischæmia does not occur (and there is no reference to it in the animal experiments of Holden and de Schweinitz, or in the human experiments of Barabaschew[14]), the only alternative, in such a case as my own, would be to accept the view of Druault, that the amaurosis is due to a direct toxic effect of quinine on the structures

of the retina. But whether quinine does or does not cause a direct action of this kind on the cells, one must not overlook the evidence that it can influence the vision through the circulation. Apart from evidence of arterial spasm derived from animal experiments, there is the observation of a case of quinine amaurosis by Stasinski[15], in which vision improved in the recumbent and diminished in the sitting posture, and that of Zanotti[16], of a girl under treatment for malarial choroido-retinitis, who after each dose of 0·75 cg. of quinine, had diminished vision associated with pallor of the discs and narrowness of the retinal arteries.

Whatever may be the change responsible for the amaurosis, we are safe in saying that it is not one of those visible with the ophthalmoscope, and if it is situated in the retina it must be microscopic. We can go further and say that while at first all the cells are so profoundly affected as to cause entire loss of vision, yet in at least a proportion of them there is complete recovery; in other words, that there may be inhibition of the function of the cells without their complete destruction.

At the best, our knowledge of the pathology of the condition is incomplete, for the researches of Holden, de Schweinitz, and Druault have not differentiated this initial, transient, change, from the permanent signs of degeneration in the retinal ganglion cells, nor have they explained the difference in behaviour of the central and peripheral parts of the retina. Again, they do not help us to understand the cloudy retinal opacity and red foveal spot which are sometimes seen. From the strong resemblance that the latter bear to the appearances resulting from embolism of the central retinal artery, it is natural to conclude that in this case also the condition of the fundus represents a coagulation-necrosis due to ischæmia. There is, however, a striking contrast in the further progress of the two conditions, for in embolism of the central artery the picture lasts for some time, and a profound loss of vision, both central and peripheral, is the rule; while in quinine amaurosis the appearance disappears in a few hours or days, and there may be complete restoration of central vision. Perhaps the difference is entirely one of degree, the arterial obstruction in the one case being due to spasm which passes off, while in the other the blockage of the central artery causes a much more complete and prolonged interference with the nutrition of the retinal elements concerned. It is evident that the retinal cloud is not, in either of the cases, the *cause* of the visual defect, since the latter can exist in the absence of retinal opacity, or persist in spite of its disappearance.

As regards the permanent fundus changes, Ward Holden, in his experiments on dogs, found atrophy of the optic nerve fibres, which he believed to be an ascending atrophy due to retinal degeneration. Nerve atrophy has also been found by others, and it appears to be

generally assumed that the persisting disc pallor in the human cases represents a true atrophy of the optic nerve. We have to remember, however, that these researches were performed on dogs, which are particularly susceptible to the effects of quinine, and that the doses given were never far short of the lethal dose. We cannot be sure that similar conditions hold good for the human eye, for opportunities for histological examination in the human subject are very rare. I have found only one such observation on record, namely, by Fortunati[17], where the patient died on the eighth day; and in this case chromatolysis of the retinal ganglion cells was found, but nothing abnormal in the optic nerves. In the case which I have reported above, the blanching of the disc and the constriction of the retinal arteries came on rapidly and simultaneously, as if there were a simultaneous constriction of all the ocular vessels. This occurred on the eighth or ninth day after the onset of blindness, and it was not accompanied by a reduction in the amount of vision or in the extent of the field; on the contrary, vision began to return twenty-four hours later, and improved for some time, while the fundus changes became only the more distinct. In view of this sequence of events it seems to me that the pallor of the disc is to be explained as the result of contraction of its blood-vessels simultaneously with contraction of those of the retina. The discs are still pale, seven months after the onset of the amaurosis, but I should hesitate to describe them as atrophic.

Several explanations might be offered to account for the better recovery of central than of peripheral vision. In the first place, it might be supposed that the initial ischæmia, assuming that to be the cause of the amaurosis, led to a more profound and permanent loss of function in the ganglion cells of the less vascular periphery of the retina. Here we are met with the difficulty which confronts all vascular explanations of the affection, namely, that there is no parallelism between the degree of vascular contraction and the amount of visual loss, for in the presence of a stationary, or even increasing, ischæmia, the vision may be steadily improving. Nor does the analogy of embolism of the central artery assist us at all, since, in a few cases of the latter which regain some vision, it is sometimes peripheral, sometimes central, sometimes in sectors, apparently according to peculiarities of the vascular supply. Again, it has been suggested that quinine exercises a selective action on the ganglionic elements in the periphery of the retina, just as tobacco selects those of the macular region. On the other hand, if we are to accept the theory that quinine acts by a direct toxic effect on the retina, through the blood stream, we must assume that the rods and cones, deriving their nutrition from the rich vascular supply of the choroid, are at least equally exposed to the poison. The different distribution, and the structural and functional differences of the rods

and cones, makes it at least possible that we have an explanation of the peripheral defect in a selective action of quinine on the rods. The night-blindness, so often complained of in these cases, supports this view; but although I suggest this as an alternative explanation, I am not aware that any histological changes have been found either to confirm or to contradict it.*

To summarise what has been said, I think that a consideration of the subject in the light of the case here reported entitles us to conclude:

1. That in quinine poisoning complete loss of vision may be found in association with a normal condition of the fundus oculi, and that there may be a striking recovery of vision in spite of the presence of well-marked fundus changes.

2. That in all, or nearly all, cases of quinine amaurosis, ophthalmoscopic changes, such as congestion of optic nerve and retina, pallor of the disc, narrowness of the retinal vessels, and cloudy opacity of the retina, make their appearance sooner or later, but that there is no correspondence between the character or severity of these changes and the intensity of the visual defect.

3. That the visual defect cannot, therefore, be due to such changes, but rather to a condition of the retinal elements invisible with the ophthalmoscope.

4. That this change may be induced, or aggravated, in the first place, by ischæmia due to contraction of the vessels of the optic nerve and retina, but that it is, in the main, the result of a direct toxic action of quinine upon the retina itself, and that the ultimate recovery of central vision, with loss of peripheral vision, and failure of vision in twilight, suggests a selective action of the poison upon the rods.

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* Dr. F. W. Edridge-Green, with whom I have had an opportunity of discussing this case, supports the suggestion here offered, and points out that if quinine inhibits, and to some extent permanently destroys, the function of the rods in the production of the visual purple, his theory of vision would lead us to expect just such a visual result in quinine poisoning as that described. A. J. B.

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THE INFLUENCE OF VASCULAR DISEASE IN THE RETINA ON THE PROGNOSIS AS REGARDS LIFE.*

BY

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THE most numerous cases of general medical interest that we, as ophthalmic surgeons, see are those that have some form or other of disease of the vascular system. It is by no means unusual for the failure of vision to be the first symptom that obtrudes itself on the notice of the patient, and sends him to seek advice of the eye specialist. These cases one examines and finds various pathological manifestations in the retina, returns them to their general medical adviser for treatment, and not infrequently loses sight of them henceforth, or else they attend the hospital, are put on to potassium iodide, and attend for a variable length of time, and then are no more seen.

With the idea of finding out what happens to these patients and when, I have undertaken an enquiry and endeavoured to trace the end of such cases. When I began, I imagined the majority of them would be dead, and accordingly first applied to the various registrars for details, but meeting with somewhat poor results, I next sent reply-paid postcards to the patients themselves, enquiring after their health, and was very agreeably surprised to find the number who were still living.

The types of retinal lesions I selected were flame-shaped hæmorrhages and signs of vascular disease in the retina, venous thrombosis and hæmorrhagic retinitis, "embolism" of the central artery, retinitis circinata; in fact, all those conditions usually associated with the condition of arterio-sclerosis. I purposely omitted true albuminuric retinitis, as the fate of such patients is well-known. A few cases of diabetic retinitis have crept in accidentally and a few traumatic hæmorrhages, but these latter have not been included in the statistics.

In one way or another I have collected 159 cases from the

* Read at the Oxford Ophthalmological Congress, July, 1916.

records of the Oxford Eye Hospital and my private records, and have managed to trace 124 of them; of the total number 63 were males, the remainder 96 females—58 of them married and 38 single.

To examine more accurately the prognostic influence of these lesions, I have arranged the cases according to their ages, taking, first, cases under 50, the majority being between 40 and 50; then, cases between 50 and 60; then those between 60 and 70; next, those between 70 and 80; and, lastly, those over 80.

Considering the first group, cases under 50 years of age. These number 26, and of these 18 are women and 8 men.

One finds a remarkable difference in the length of life in the various cases, and this difference is seen to be due to the presence of albumen in the urine.

For example—of those with albumen present:

Two died after five years, one of cerebral hæmorrhage, the other of phthisis.

One died after three years ten months, of chronic Bright's disease.

One „ three „ five „ , of nephritis.

One alive after two „ three „

One died in three months, of granular kidney.

One „ four „ , a syphilitic subject.

One alive after six years, in spite of a blood pressure of 220 mm., but comparing these with those that had no albumen, one finds:—

One living after 28 years. Age 74.

„ „ „ 25 „ Age 40. (Case of embolism.)

„ „ „ 17 „ Age 66.

„ died „ 16 „ of cerebral thrombosis, pneumonia, and heart failure, at the age of 67, but he had four strokes.

One living after 15 years. Age 50.

„ „ „ 11 „ Age 55.

„ „ „ 4 „ Age 48.

In many of the cases I, unfortunately, have no note as to the state of the urine; but one has enough evidence, I think, to say quite definitely that in young patients with vascular disease of the retina, the presence or absence of albumen in the urine is by far the most important factor in forming an opinion as to the prognosis *re* life.

Taking next the cases between 50 and 59. They number 47, of whom 28 are women and 19 men.

The difference in prognosis between those with and without albumen is not so marked as in the preceding group. Whereas in that group the longest life I could find with albumen was six years, many in this one have lived a considerable time. I need not weary you with reciting the whole number, but just refer to a few, such as Case No. 64. At the age of 58 had flame-shaped hæmorrhages in the left eye, and some albumen in the urine. She is still alive, age 71

—13 years after the original lesion. Another lived nine years, dying of syncope, age 63. She had hæmorrhagic retinitis and thickened arteries.

Another case of interest is No. 67—a man who had a vitreous hæmorrhage, arteries enormously thick, blood pressure of 200, and a good deal of albumen in the urine. When I first saw him, eighteen months ago, he was quite broken down, could not manage his business, and was altogether in a most tottery condition. I saw him again a month ago. His vitreous hæmorrhage had practically cleared up; he was altogether better and brighter in himself; and he had had a severe attack of appendicitis, with bad operation, in the meantime.

Of cases without albumen, two died under two years; one of them (No. 46) had a blood pressure of 200 mm. and died of a stroke. Others died at 4, 5, 6, and 12 years respectively. Many of the rest are alive at times varying from 1½ to 16 years, the latter being a woman who had extensive retinal hæmorrhage at the age of 54, and who is still alive, age 70; although she is beaten by Case No. 42, a man with hæmorrhagic retinitis at the age of 54, but no albumen, who is still alive, age 76; 22 years after the original lesion.

Passing on to those cases developing between 60 and 69, they number 54, and are evenly divided, 27 females and 27 males. As before, cases with albumen compare unfavourably with those without.

Examples of cases with albumen:

One died in four years with cardiac asthma.

One „ six months with a stroke.

One living after three and a half years, but in poor health.

One „ two years, age 66.

One „ three and a half years, age 68.

Comparing these with those known to have had no albumen:

(114) Retinitis circinata R.E. at the age of 67. Retinitis circinata L.E. at the age of 66 and much hæmorrhage. Still alive and well after 10 years. Another died after six years, age 70.

(80) Had a blood pressure of 250 at the age of 64, with flame-shaped hæmorrhages. Died four and a half years later, old hemiplegia and acute bronchitis.

(84) Had hæmorrhages in the left eye followed by glaucoma twenty-five years ago, and is still alive, age 84.

(85) Another at 65 years, who had venous thrombosis, died of senile decay, aged 81, sixteen years later.

(91) At 68 years, thickened arteries and ? embolism, died at the age of 78 of cerebral hæmorrhage, about ten years later; and so on.

Thus at this age the prognosis *re* life of retinal vascular disease

is, on the whole, extraordinarily good, the normal expectation of life at 60 being roughly 13½ years, and at 69 nearly 9 years.

Now coming to the cases between 70 and 80. These number 26; 20 females and 6 males. They show the remarkable age to which these patients live.

(129) Age 75, male, with much albumen. Hæmorrhages and thickened arteries, is still alive, age 83, after 8 years.

Other cases are:—Alive, age 75, after 2 years; 77, after 5 years; died at 83, after 10 years; died at 79, after 8 years; alive at 75, after 5 years.

(136) Alive at 80, living 8 years and 3 months.

(139) Alive at 80, after 6 years.

(143) Blood pressure 220, no albumen, died aged 78, of a cerebral hæmorrhage, living 5½ years.

(145) Hæmorrhage at age of 75, alive at 82, after 7 years.

(149a) Vascular disease and diabetic retinitis, alive after 5 years, age 78.

One other case I forgot to mention is a man (No. 75) who had both albumen and sugar at the age of 60, and who lived for 7 years before his death, the actual cause of which I could not discover.

Finally, those 80 years of age and over.

There are 5 of these cases—3 men and 2 women.

(151) Age 87, with hæmorrhage, died, aged 93, of senile decay.

(152) Age 81, with albumen and hæmorrhage, still alive, aged 86.

(153) Age 86, embolism, died, aged 89, of cerebral hæmorrhage.

(154) Age 80, hæmorrhage and thickened arteries, still alive, aged 84.

The fifth I cannot trace after seven months, but these few cases bear out the fact, I think, that the older the patient, the less important from the point of view of prognosis to life are the vascular diseases of the retina.

As regards the treatment of these cases, this, of course, is the treatment of their general condition. Rules for their course of life, and instructions as regards diet, are important, with the idea of giving their kidneys as light work as possible; but perhaps the most important is, to avoid all physical strain.

As regards use of the eyes, I should like to hear the opinion of others. Whilst it is common-sense not to work a damaged organ hard, I must confess I have never seen any damage done to these eyes by ordinary use; and as it is a condition which may last more or less to the end of the patient's life, it seems to me a mistake to prohibit the use of the eyes, as is sometimes done, for, after all, what is the use of having eyes if they are never used.

Lastly, as regards causation. Very many are no doubt due to thrombosis of vein or artery, and in some cases it is difficult to

determine exactlv which vessel is at fault. Some years back, after Sir Almroth Wright's suggestion, that many ocular hæmorrhages might be due to thrombosis, I measured the coagulation-time of the blood, but, as a rule, rarely found much variation from the normal. Nor is this, I think, to be wondered at. The lesion may have occurred a few days to a few months before being seen, and the coagulation-time is known to vary very rapidly with differences in diet, time of day, length of time after meals, etc. The only thing of interest I did come across was the fact that potassium iodide had by far the most powerful action in prolonging the coagulation time of any drug I tried. In my experience, it was much more potent in this respect than citric acid, and in those cases of iodism where the symptoms are almost if not entirely due to this property, they can, to a certain extent, be obviated by giving calcium at the same time, or, what comes to the same thing, giving it in large doses of milk, as one used to be taught some years before anyone took any notice of coagulation-times.

This fact, in addition to its power of lowering the blood pressure, makes the drug of real value in the treatment. Other means of lowering the blood pressure, such as electrical treatment, I have been disappointed in; the effect seems to be only temporary, and blood pressure, after all, is but a symptom, and one that the patient does not object to. One old man, in particular, I remember used to say, " I'm sure my blood pressure is well over 200; I feel so fit and energetic."

Final conclusions from study of statistics.—Retinal lesions are considerably more common in women than men, and that not altogether due to child-bearing, as 38 of the 96 women were, as far as I could discover, unmarried. The cases were most numerous between the ages of 60 and 70, and next between 50 and 60.

The older the patient, the better the prognosis as regards life, irrespective of the presence of albumen in the urine to a large extent, whilst the younger the patient, the worse the prognosis, especially so if albumen is present in the urine.

Lastly, I wish to thank Dr. A. L. Ormerod, M.O.H. for the City of Oxford, and Dr. E. Morton, M.O.H. for part of the County of Oxford, for their valuable advice and assistance in the tracing of many of the cases.

Number and Initials and Sex.	Reference Number.	Age and First Attendance.	Diagnosis. First Hæmorrhages.	Age and Last Attendance.	Duration of Life.	Age at and Date of Death.	Cause of Death.
1 J. C. (M)	A 24910	48—May 22, 1912	Stroke 18/12 ago. Many hæmorrhages. Vascular disease	48—May, 1912	3/12	48—13 Aug., 1912	Granular kidney. Cerebral irritation. Exhaustion
2 M. H. (F)	A 18124	49—July, 1909	Few small hæmorrhages in both. Albumen. No sugar	July, 1911	5 years	54—July 18, 1914	Cerebral hæmorrhage (35 days)
3 D. D. (F) S.	A 29807	25—Feb., 1914	Hæmorrhage at macula. Myopia.	May, 1914	2½ years	27—Alive	—
4 A. Y. (M)	A 33562	39—June 14, 1915	Retinal hæmorrhage, traumatic.	Jan., 1916	1 year	40—Alive	—
5 L. B. (F) S.	A 27487	16—April 9, 1913	Coag. time, 3 h. 3½ min. Anæmia. Neuro-retinitis. Hæmorrhages. B.P. 140	June, 1914	—	No trace	—
6 A. E. (M)	A 21519	23—Jan. 12, 1911	Traumatic hæmorrhage. R. retina. Albumen in urine	March, 1911	2 years 4/12	May, 1913	Uræmia
7 H. S. (F) Md.	12661	49—April, 1896	2 weeks. Hæmorrhagic retinitis. Probably venous thrombosis L.	63—August, 1908	16 years	67—June 30, 1912	Pneumonia. Cerebral thrombosis and heart failure
	A 16104	63—Aug., 1908	4 strokes				
8 C. A. (M)	A 933	46—March, 1888	Hæmorrhages R. macula. Urine 1009. No albumen. No sugar. Slight optic neuritis R.E. Hæmorrhage L.E.	60—Sept., 1902	28 years	Alive	—
9 R. J. K. (M)	A 25562	38—Aug. 16, 1912	Thick arteries. Hæmorrhage in both. B.P. 190. Syphilitic. Hemianopsia.	Dec. 1912	4 months	Dec., 1912	Cerebral softening, occipital lobe. Chronic endocarditis, etc. Arteries thickened.
10 H. S. (M)	6491	27—Nov., 1891	Hæmorrhage in retina. No albumen or sugar	40—April, 1904	25 years	Alive	—
10A E. C. (F)	A 3128	46—Nov. 28, 1901	Hæmorrhages and flecks R.E. Albumen in urine	46—Nov., 1901	3 years 10/12	Sept. 6, 1905	Chronic Bright's disease. Uræmia
11 M. A. B. (F) Md.	A 9438	44—March, 1905	Hæmorrhages in retina L. No albumen. No sugar	55—April, 1916	11 years	Alive	—

12 R. C. (F) S.	A 27305	44—Feb., 1910	Hæmorrhages in retina. Gross arterio-sclerosis. Albumen. B.P. 210-220	50—May, 1916	6 years	Alive	—
13 M. S. (F)	A 1181	43—Oct. 3, 1900	Embolism of central artery L.	Dec., 1900	7 3/12	No trace	—
14 F. H. (M)	A 1655	35—Jan. 23, 1901	L. E. hæmorrhages in retina. Arterial thrombosis. No albumen or sugar	35—May, 1904	15 years 5/12	Alive	—
15 M. A. H. (F)	A 13674	47—May 29, 1907	Hæmorrhages in retina R. E. Arteries + +. Albumen in urine	47—Oct., 1907	3 years 5/12	51—Oct. 31, 1910	Chronic nephritis
16 E. S. (F)	A 30795	48—June 10, 1914	Retinal hæmorrhage L.E. No albumen. No sugar	48—June, 1914	2 years	50—Alive	—
17 E. S. (F) S.	A 10723	44—Jan. 19, 1895	Embolism of central artery L.	Sept., 1895	7 9/12	No trace	—
18 L. W. (F) Md.	16888	44—Dec. 14, 1898	Hæmorrhage L. macula. No albumen.	Nov., 1902	4 years	No trace	—
19 A. A. S. (F) Md.	A 30216	44—Mar. 25, 1914	Hæmorrhages in both. Albumen.	45—April, 1915	1½	?	—
20 J. B. (M)	A 3932	38—May 21, 1902	Hæmorrhages in both. No albumen. No sugar	April, 1903	1 year ?	No trace	—
20A I. T. (F) Md.	13779	49—Nov. 25, 1896	White flecks in R. retina. No albumen. May 1913. Recent hæmorrhages	66—Dec., 1913	17 years	No trace	—
21 H. B. (F) Md.	A 2065	48—Apl. 24, 1901	3/12 Hæmorrhages in both. Albumen	April, 1901	5 years	53—Jan. 20, 1906	Phthisis
22 P. W. (M)	A 26433	24—Nov., 1912	Traumatic hæmorrhages	Jan., 1913	3½ years	Alive	—
23 A. L. (F)	8445	19—May, 1893	Embolism central artery	22—Feb., 1896	21 years	Alive	—
24 E. B. (F) Md.	A 31072	41—July, 1914 9/12 Hist	B.P. 270. Arteries bright. Patches of œdema. Hæmorrhages.	Sept., 1914	1 year	41—Nov., 1914	Chr. nephritis, parenchym.
25 N. W. (F) Md.	A 30205	37—March, 1914	Arterio-sclerosis. Hæmorrhages in retinæ. High tension and albumen	July, 1914	2 years	Alive	—
26 E. C. (F) Md.	A 34363	41—Sept., 1915	Hæmorrhages in both retinæ.	Sept., 1915	9/12	Alive	—

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27 G. B. (M)	A 31478	50—Sept., 1914	Hæmorrhage, left retina	50—Sept., 1914	1 year 9/12	52—Alive	—
28 E. D. (F)	A 7698	50—May 7, 1913	Arterio-sclerosis and hæmorrhages in both eyes. B.P. 240. Albumen	51—Feb., 1914	3/12	Alive	—
29 W. T. (M)	13367	53—Aug. 26, 1896	Retinitis hæmorrhagica. Aortic disease	53—August, 1896	—	—	—
30 E. H. (F)	A 28717	52—Sept.. 1913	2/12. Hæmorrhage. Right macula.	52—October, 1913	29/12	54—Alive	—
31 R. W. (F)	A 9813	52—May, 17, 1905	2 weeks. Venous thrombosis R.	52—July, 1907	11 years	63—Alive	—
32 G. M. (M)	11237	52—May, 15, 1895	3 weeks. Plugging arteria centralis	July, 1896	5 years 5/12	57—Oct. 13, 1900	Softening of the brain. Syncope
33 E. N. (M)	P. P.	53—Nov., 1905	Retinitis circinata	April, 1914	11 years	64—Alive and well	—
34 A. W. (M)	P. P.	53—March, 1914	Partial thrombosis of vein. Arteries +. B.P. 180. No albumen	March, 1914	2½ years	55—Cardiac asthma	—
35 A. H. (F) Md.	A 36785	53—Oct. 13, 1914	Partial venous thrombosis. Arteries +. Flame-shaped hæmorrhages	June, 1916	1 year 9/12	56—Alive and fairly well	—
36 J. (F)	A 6944	51—July, 1915 Aug. 1915	Hæmorrhage in L. E. White flecks at macula L. B.P. 180. Trace of albumen occasionally	—	1 year	Alive and well	—
37 G. B. (M)	15784	53—May, 1898	Hæmorrhagic retinitis. No albumen. Aortic disease	May, 1898	4 years	57—1902	?
38 M. J. (F)	A 62	54—January, 1900	Venous thrombosis. No albumen or sugar	March, 1900	3/12	No trace	—
39 A. P. C. (F) Md.	A 714	54—July 4, 1900	3/7. Hæmorrhages in retina R.E. extensive	Dec., 1902	16 years	70—Alive	—
39a J. H. (M)	A 12516 A 4254	58—July 30, 1902	1 year R. retinal hæmorrhages. No albumen. No sugar	Oct., 1906	12 years	68—Jan. 25, 1913	Cerebral softening, arterio-sclerosis, cerebral hæmorrhages

40 S. S. (F)	A 8791	54—Oct. 31, 1904	3 days. Embolism central artery L.	54—July, 1907	9 years	63—Nov. 6, 1913	Bright's disease. Morbus cordis
41 M. M. (F) Md.	A 17384	53—Mar. 24, 1909	Hæmorrhages R.E. Arteries ++. No albumen. No sugar. Sp. gr. 1015	July, 1907 May, 1916	7 years	Alive and pretty well	—
42 H. C. (M)	9607	54—April, 1894 70— 1910	Hæmorrhagic retinitis R.E. No albumen. No sugar.	May, 1911	22 years	Alive	—
43 H. B. (F) Md.	A 19808	53—Apr. 20, 1910	2/12. Gross hæmorrhages in R.E. Older hæmorrhages in L.E. Albumen in urine	54—Feb., 1911	1 year	Feb. 23, 1911	Mitral regurgitation. Syncope
44 J. W. (M)	A 17414	54—March, 1909 57—August, 1912	2/7. Venous thrombosis L.E. No albumen. No sugar. 5 days R.E. went. Œdema. Hæmorrhages.	Sept., 1912	7 years	Alive	—
45 E. L. (F) Md.	17418	54—April, 1899	Hæmorrhagic retinitis R. Arteries +. Trace of albumen.	60—August, 1905	9 years	63—Dec. 28, 1908	Syncope, heart failure
46 H. (M)	A 5276	54—Oct. 2, 1913	Much vascular disease of retina. Small flame hæmorrhage. B.P. 200. No albumen.	Nov., 1913	1 year 3/12	January, 1915	Cerebral hæmorrhage
47 E. S. (F.) Md.	2927	54—Sept., 1889	Choroiditis. Neuro-retinitis.	65—Nov., 1898	8 years	72— 1905	?
48 G. C. (M)	A 27197	54—Mar. 15, 1913	6/12. Hæmorrhage both retinæ.	54—May, 1913	1 year 6/12	March, 1914	?
49 J. W. (F) Md.	18812	55—Nov., 1909	Hæmorrhage L. macula. No albumen. No sugar	55——June, 1911	1 year 7/12	?	?
50 M. A.W. (F) Md.	A 24694	52—Apl. 24, 1912	Fine hæmorrhages L.E. Sugar in urine	52—April, 1912	1 year 8/12	54—Dec. 13, 1914	Diabetes
51 E. R. (F)	15359	54—Feb. 16, 1898	Hæmorrhagic retinitis R. Urine, no albumen. Aortic disease	54—Feb., 1898	—	No trace	—
52 E. S. (F) Md.	A 10331 A 23454	53—Aug. 30, 1905 59—Oct. 25, 1911	Myopia Diabetic retinitis. Sugar.	Oct., 1911	—	?	?
53 T. (F)	P. P.	54—Sept., 1913	Recent hæmorrhages R. retina. High tension pulse. B.P. 200-210	Oct., 1914	2 yrs. 9 mths.	Alive and well	—

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54 M. G. (F) Md.	A 30267	55—April, 1914	Flame-shaped hæmorrhages No albumen. No sugar	April, 1914	—	?	?
55 J. H. (F) Md.	A 32501	55—Feb. 10, 1915	Hæmorrhages R. retina. No albumen. No sugar	Sept., 1915	1 year 6/12	56—Alive	—
56 E. V. (F) Md.	A 15335	56—April, 1908	3/7. Stroke 5 years ago. Albumen. Hæmorrhages retina. Arteries + + .	Nov., 1908	4 years 1/12	60—May 26, 1912	Chronic nephritis, cardiac dilatation and failure.
57 W. H, S. (M)	11819	56—Sept., 1895	Hæmorrhages L. retina. Proliferating membrane	August, 1896	11 months	No trace	—
58 E. E. (F) Md.	A 6908	57—Nov. 25, 1903	3/7 Flame-shaped hæmorrhages R. Albumen	Sept., 1904	8 years 4/12	65—Mar. 3, 1912	?
58A S. H. (F) Md.	A 30509	56—Apl. 30, 1914	Arterio-sclerosis. Retinal hæmorrhages. Albumen. Sugar. B.P. 150-160	56—June, 1914 58—May, 1916	2 years	Alive. Fairly good health	—
59 D. A. (F) Md.	A 22848	59—Aug. 2, 1911	Vascular disease of retina and hæmorrhages. Urine 1035. No albumen. No sugar	59—August, 1911 60—April 13, 1912	8/12	No trace	—
60 M. A. K. (F) Md.	16722	56—Nov. 9, 1898	Hæmorrhages both eyes, especially L. Albumen in urine	56—Nov., 1898	No trace	—	—
61 S. H. (F)	16238	56—Aug. 3, 1898	Hæmorrhagic retinitis	56—Oct., 1898	3/12	No trace	—
62 S. S. (F)	14594	56—June 30, 1897 1900 74—July, 1913	Vitreous opacities. Arteries + +, and hæmorrhages R.E.	Oct., 1914 74—June, 1916	3 years	77—Alive and well	—
63 J. P. (M)	A 11825	59—June 6, 1906	2/12. R. retinal hæmorrhages. No albumen. No sugar. 1009. Coagulation time: 2 minutes	July, 1907	1 year 3/12	?	?
64 E. C. (F) Md	A 6255	58—Aug. 5, 1903	Retinal hæmorrhage L.E. Some albumen. No sugar	58—Oct., 1903	13 years	71—Alive	—
65 F. K. (M)	A 1678	59—Jan. 30, 1901	1/7 hæmorrhages L. retina. No albumen. Sugar present	59—April, 1901	6 years	26—Feb., 1907	Meningitis and coma —

66 B. (F)	P.P.	57—Jan., 1912 April, 1914	Hæmorrhagic retinitis	August, 1915	2 years 3/12	Alive and well	
67 W. W. (M)	A 6476 P.P.	58—Jan., 1915	Vitreous hæmorrhages R. Hæmorrhages and thick arteries L. B.P. 200. Albumen	June, 1916	1 year 6/12	Alive and well	—
68 H. (M)	A 1497 P.P.	57—August, 1908 60—August, 1911 61—Sept., 1912 64—May, 1915	Veins full. Arteries tortuous Albumen 1/6. Hæmorrhages in both. Arteries ++. Much hæmorrhage in both. B.P. 180, 200, 212	June, 1915	4 years	64—	Cardiac asthma
69 I. F. (M)	11143	58—April, 1895	Hæmorrhagic retinitis both eyes. No albumen. No sugar	July, 1895	17 years	75—1912	Stroke
70 A. C. (F)	15615	57—April, 1898 July, 1901	Venous thrombosis R. Hæmorrhage L.E.	Jan., 1903	6 years	Jan., 1904	Cerebral hæmorrhage
71 M. P. (M)	A 13656	58—May, 1907	Flame-shaped hæmorrhage L.E. No albumen. No sugar. B.P. 140	May, 1908	9 years	Alive	—
72 J. D. (M)	17231	60—Feb. 22, 1899	Hæmorrhage, R.E.	Feb., 1899	?	Cannot trace	—
73 A. B. (F) Md.	A 25633	60—Aug. 28, 1912	Subhyaloid hæmorrhage	Dec., 1912	4/12	?	—
74 E. C. (F) Md.	11494	60—July, 1898	Venous thrombosis R.	63—May, 1899	12 years	72—July 20, 1909	Chronic Bright's disease, 3 years. Uræmic convulsions, 4 months
75 H. H. (M)	A 13165	60—Feb., 1907	2/12. Albumen and sugar. Hæmorrhages in both.	61—May, 1908	7 years	67—Oct., 1914	?
76 G. B. (M)	A 25539	60—Aug. 14, 1912	Retinal hæmorrhage. Arterio-sclerosis. Urine sp. gr.: 1010. No albumen. No sugar	Sept., 1913	4 years	Alive	—
77 S. W. (M)	A 1463	60—Dec. 5, 1900	Intra-ocular hæmorrhage. No albumen. No sugar	Oct., 1903 Nov., 1904	4 years	——	—
78 E. T. (M)	A 9063	60—Dec. 31, 1904	Embolism cent. art L.	68—June, 1912	8½ years	70—June 3, 1914	—
79 M. A. W. (F) Md.	A 279	60—Mar. 28, 1900	3 weeks. Many hæmorrhages R.E. White patches No albumen.	76—August, 1900	16 years	Alive	—

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80 J. L. (M)	A 22083	64—April 12, 1911	Small flame-shaped hæmorrhages R.E. and L.E. Urine: no albumen or sugar. B.P. max. 250 mm. min. 190	April, 1911	4½ years	Nov. 22, 1915	Old hemiplegia (R.) and acute bronchitis
81 J. C. (F) Md.	A 24510	66—March, 1912	Vitreous opacities. Hæmorrhages R. No albumen or sugar	May, 1912	3/12	No trace	—
82 U. G. (M)	A 12533	67—Oct. 10, 1906	Hæmorrhage in L.E. Arteries +.	68—May, 1907	3 years 9/12	71—Jan. 25, 1910	Morbus cordis
83 T. B. (M)	A 3116	69—Oct. 27, 1901	4/7. R. embolism	Nov., 1903	2 years 3/12	70– Jan., 1904	Cerebral hæmorrhage. Coma
84 T. C. (M)	13676 A 9291	64—Nov. 1896 66— 1898	Hæmorrhage in L.E. Glaucoma, 1905	Sept., 1905	28 years	Alive	—
85 A. M. (F)	13610	65—Oct. 21, 1896	Venous thrombosis R.E.	May, 1904	16 years	81—Nov. 6, 1912	Senile decay. Heart failure
86 H. P. (F) Md.	11868	66—Oct. 2, 1895	Embolism central artery L.	Nov., 1895	4 years 9/12	70—June 20, 1900	Cerebral hæmorrhage
87 J. G. (M)	A 25341	66—July, 1912	Chronic glaucoma R.E. Retinitis. Hæmorrhages. No albumen. No sugar	August. 1912	4 years	Alive	—
88 J. A. (F) Md.	7159	62—1892	March, 1898. Aged 68. Hæmorrhagic retinitis	March, 1898	7 years	75—Aug. 13, 1905	Cystitis (10 years) and uræmia. Exhaustion (3 days)
89 E. G. (F)	A 26095	63—Oct. 23, 1912	Retinal hæmorrhages. Arterio-sclerosis. Flame-shaped hæmorrhage L.E. No albumen. No sugar	Jan., 1916 April, 1916	4 years	66—June 18, 1916	Cerebral hæmorrhage
90 E. C. (F) Md.	A 1182	65—Oct., 1900	Embolism central artery. No albumen	65—Oct., 1900	8 years	73—Feb. 28, 1908	Morbus cordis
91 S. C. (F) Md.	A 8601	68—Sept., 1904	1/7. Heart-sounds normal. Arteries thick. ? Embolism	68—Nov., 1904	10 years 5/12	78—Feb. 25, 1915	Cerebral hæmorrhage. Coma

92 S. K. (M)	A 15058	68—Feb., 1908	Hæmorrhage L. macula. Coag. 1·45 B.P. max. 149, min. 95	Aug., 1913	6 years 9/12	75—Nov. 26, 1914	Stroke
93 A. C. (F) Md.	A 3965	65—May, 1902	Hæmorrhagic retinitis. No albumen. No sugar	May, 1903	1 year	No trace	—
94 J. C. (M)	A 309	68—April, 1900	Hæmorrhagic retinitis	Oct., 1900	6/12	No trace	—
95 E. C. (F) Md.	A 31188	65—Aug. 5, 1914	Had 2 strokes. Albumen. Few hæmorrhages	Aug., 1914	2 years	Alive, not very well	—
96 A. R. (F) Md.	A 9207 A 17498	67—Feb., 1905 71—April, 1909	Hæmorrhages L. retina. Vitreous hæmorrhage	April, 1909	7 years	74—Mar. 20, 1912	Heart failure and old age
97 E. A. (F) M .	A 21371	69—Dec. 21, 1910	Hæmorrhages in both. Arteries thick. B.P. 122 max. 105 min. Urine, no albumen, no sugar	73—Sept., 1915	6 years	Alive	—
98 O. C. (F)	A 6939	64—Jan. 9, 1915	Thrombosis of branch of retinal vein and hæmorrhages L.E. No albumen	Nov., 1915	1 year 7/12	Alive and well	—
99 J. R. (M)	10511	63—Nov., 1894	L.E. hæmorrhagic retinitis. No albumen or sugar	65—Jan., 1896	2 years	Dead	?
100 F. P. (M)	A 661	61—June 27, 1900	7/7. Venous thrombosis	64—Nov., 1903	8 years 3/12	69—July, 1908	Cerebral hæmorrhage
101 E. S. (F) Md.	16643	65—Oct. 26, 1898	Hæmorrhagic retinitis, white flecks in both	65—Nov., 1898	7 years	72—Dec., 1905	?
102 D. (F) Md.	A 31041	64—July, 1914	Hæmorrhages. Vascular disease. Albumen	July, 1914	2 years	Alive	—
103 W. F. (M)	A 34674	65—Oct. 23, 1915	6 days. Thick vessels. Hæmorrhages right retina. Albumen in urine. Urine, 1020	65—Nov., 1915	6/12	27—March, 1916	Stroke
104 M. W. (F) Md.	A 31258	62—Aug. 19, 1914	Hæmorrhages both. No albumen. No sugar	62—August, 1916	1 year 10/12	Alive	—
105 E. B. (F)	A 5672	64—April 22 1903	Retinal hæmorrhage R.E.	64—April, 1903	1 week	25—April, 1903	Pneumonia and heart failure
106 W. G. (M)	A 28200	65—July 9, 1913	Retinal hæmorrhage. Lead poison. No albumen. No sugar	65—April, 1914	2 years	67—August, 1915	Heart failure

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107 J. P. (M)	15337	64—Feb. 9, 1898	Venous thrombosis. No albumen. Ht. sound.	64—Nov., 1898	6 years	70—1914	?
108 M. P. (F)	A 9281	64—Feb. 8, 1905	July, 1912. Retinal hæmorrhages R. Age 71	74—August, 1914	4 years	75—Alive	—
109 J. P. (M)	A 20915	62—Oct., 1910	Hæmorrhages R. retinal. B.P. 125 max., 105 min. Some albumen	63—May, 1911	4 years	1914	—
110 J. W. (M)	A 22463	68—June 7, 1911	6 weeks. Large retinal hæmorrhages. Arterio-sclerosis. No albumen. No sugar	August, 1911	4 years	72—April 4, 1915	Broncho-pneumonia and heart failure
111 G. P. (M)	10688	61—Jan. 2, 1895	3 weeks. L. many hæmorrhages	Jan., 1895	——	No trace	—
112 D. C. (M)	A 7973	69—June, 1904	2/7. L. embolism. central artery. Slow pulse	69—July, 1907	7 years	76—June 12, 1911	Morbus cordis. Bronchitis. Asthenia
113 J. (M)	A 6482	61—Nov., 1912	Flame - shaped hæmorrhages in both eyes. Albumen in urine	June, 1916	3½ years	Alive. Poor health. R.E. gone. L.E hæmorrhages	—
114 D. (F) Md.	A 6686 P P	67—1906 76—1915	Retinitis circinata R.E. Retinitis circinata L.E. and much hæmorrhage	June, 1916	10 years	Alive and well	—
115 M. (F) Md.	A 7572	65—May, 1914 66— ,, 1915	Arteries bright. Thrombosis of branch of retinal vein. B.P. 200. No albumen	June, 1916	2 years	Alive and well	—
116 G. T. (F.)	A 15382	68—April, 1908	Flame - shaped hæmorrhages, L.E. Dropsy	May, 1908	1 year, 6/12	69—October, 1909	Dilated, fatty heart. Bronchitis
117 G. B. (M.)	A 2079	66—April, 1901	Diabetic retinitis	——	2 years	1903	—
118 E. W. (F)	A 162	67—Dec., 1893 Choroiditis R.	74—Hæmorrhagic retinitis R.E. Feb., 1900	Aug., 1901	8 years 18 months	81—1908	Heart disease
119 E. C. (F) Md	10599	63—May, 1889	67—Dec. 1894. Hæmorrhages in retina	Jan., 1895	5 years	69—1900	?

120 I. P. (M)	11548	64—July, 1895	Punctate hæmorrhage, R. Venous thrombosis L. Albumen, trace	Nov., 1895	5 months	?	?
121 T. C. (F) Md.	A 6207	65—July, 1903	Thrombosis vein L.E. Vitreous hæmorrhages R. No albumen	Sept., 1903	7 months	66—January, 1904	?
122 A. M. (F)	A 15829	64—July, 1908	Retinal hæmorrhages. No albumen or sugar. B.P. 185-135	August, 1911	7 yrs. 4 mnts.	71—Nov., 1915	Influenza
123 B. (M)	A 31499	62—Sept., 1914	Hæmorrhages in retina. No albumen or sugar	Sept., 1914	2 years	Alive	—
124 J. J. (M)	A 33942	69—July, 1914 10 months' history	Retinal hæmorrhages. Chronic glaucoma	Still attending	1 year	Alive	—
125 J. T. (M)	A 26685	68—Jan., 1913	Hæmorrhages in retina. L.E. arteries thick. Albumen in urine.	Sept., 1913	3½ years	Alive	—
126 E. P, (F) Md.	A 7162	72—Jan. 13, 1904	1/12. Hæmorrhages R.E.	January, 1904	?	No trace	—
127 E. S. (F)	A 29974	75—Feb. 25, 1914	Hæmorrhage L.E.	77—Feb., 1916	2 years, 4/12	77—Alive	—
128 M. E. (F) Md.	A 3908	76—May 14, 1902	Venous thrombosis L.	76—Aug. 13, 1902	?	No trace	—
129 W. W. (M).	A 15913	75—July, 22, 1908	Arteries + +. Hæmorrhage ? Embolism. Much albumen	August, 1908	8 years	Alive	—
130 S. R. (F) Md.	A 30790	73—June 10, 1914	Chr. Glaucoma. Hæmorrhage L.E.	August, 1914	2 years	Alive	—
131 E. G. (F)	A 22578	72—June, 1911 August, 1913	Albumen in urine. Hæmorrhage in R.E. Arteries +. Hæmorrhage in both	August, 1913	5 years	Alive	—
132 H. R. (F) Md	14972	73—Sept., 1897	Hæmorrhage in R.E.	October, 1897	10 years	83—Mar. 12, 1907	Bronchitis. Asthenia
133 M. C. (F)	18309	70—Oct. 4, 1899	3/7. Hæmorrhage in ret. Sugar in urine	Nov., 1899	7 years	77—Aug. 2, 1906	Senile decay
134 S. H. (F)	A 22751	70—July 19, 1911	Neuro-retinitis. Hæmorrhage. No albumen. No sugar	October, 1915 May, 1916	5 years	Alive	—

Number and Initials and Sex.	Reference Number.	Age and First Attendance.	Diagnosis. First Hæmorrhages.	Age and Last Attendance	Duration of Life.	Age at and Date of Death.	Cause of Death.
135 J. V. (F) Md.	17751	76—June 7, 1899	Hæmorrhage R.E.	76—June, 1899	?	No trace	
136 M. S. (F) Md.	A 15788	72—Mar. 19, 1908	5 days. Hæmorrhage right retina. Urine, no albumen, no sugar	72—Sept., 1910	8 years 3/12	80—Alive	—
137 E. P. (F) Md.	A 32824	74—Mar. 17, 1915	Hæmorrhage right retina	74—June, 1915	1 year 3/12	75—Alive	—
138 C. K. (M)	A 6232	77—July 29, 1903	Embolism art. cent. R.	77—August, 1903	?	Dead	?
139 K. N. (F)	A 21489	74—Jan. 11, 1911	6/12. Retinal hæmorrhage both. B.P. 197. No albumen. No sugar. Sp. gr. 1005 Arteries +.	July, 1911	6 years	80—Alive	—
140 Z. C. (F) Md.	16832	72—Nov., 1898	3/12. Hæmorrhagic retinitis	Nov., 1898	6 years	78—Died 1904	Unknown
141 A. P. (F)	17086	79—Jan. 18, 1899	Hæmorrhages in both eyes.	Jan., 1899	—	Dead	Unknown
142 M. S. (F)	A 30882	72—June 24, 1914	Hæmorrhages in both. High tension pulse. Third nerve paralysis	July, 1914	—	No trace	—
143 M. C. (F) Md	A 18764	72—Nov., 1909	Retinal hæmorrhage R. B.P. 220 max. 140 min. No albumen. No sugar	78	5½ years	78—April 11, 1915	Cerebral hæmorrhage, coma
144 L. (F)	A 6942	70—July 12, 1915	Hæmorrhages both retinæ	Aug., 1915	1 year	71—Alive and well	—
145 F. K. (M)	A 18178	75—July, 1909	Hæmorrhages in retina. No albumen. No sugar	—	7 years	82—Alive	—
146 L. S. (F) Md.	13692	70—Nov., 1896	Hæmorrhages in retina	7 —June, 1899	3 years ?	?	?
147 I. J. (M)	18253	71—Sept., 1899	Embolism central artery	Dec., 1899	—	—	Stroke
148 B. (M)	A 8449	74—Aug., 1904	Hæmorrhages in retina. Faint albumen. No sugar. Pulse, high tension. Four strokes	76—July, 1906	3 years	76—Feb., 1907	Cerebral hæmorrhages

149	H. M. (M)	16097	70—Sept., 1891 No hæmorrhage	Spots of hæmorrhage. Dec., 1897	May, 1901 Hæms., age 80	7 years	83—1904	?
149A	E. M. (F) Md.	A 22868	71—Aug., 1911	Vascular disease. Diabetic retinitis	72—May, 1912	5 years	Alive	—
150	E. M. (F) Md.	16359	75—August, 1898	Hæmorrhages R. retina	76—Feb. 1899	6 months	No trace	—
151	G. T. (M)	A 20639	87—Aug. 24, 1910	Hæmorrhages R. retina. No albumen. No sugar	87—Oct., 1910	5 years 8/12	93—April 27, 1916	Senile decay
152	S. A. (F) Md.	A 23220	81—Sept. 20, 1911	5/7. Hæmorrhage L. E. Trace of albumen	Nov., 1911	5 years	Alive	—
153	J. G. (M)	15749	86—May 11, 1898	Embolism art. centralis R.E.	August, 1898	3 years 6/12	89—Dec., 1901	Cerebral hæmorrhage
154	J. H. (M)	A 24768	80—May 1, 1912	3/7. Hæmorrhages in retina L.E. High T. pulse. Arteries + .	80—May, 1912	4 years 2/12	Alive	—
155	A. R. (F)	A 14864	87—Jan., 1908	Hæmorrhages	July, 1908	7 months	?	?
156	C. B. (F)	A 33887	62—July, 1915	Retinal hæmorrhages. Arterio-sclerosis. Trace of albumen	Aug., 1915	1 year	Alive	—

NOTES.—M = male.
F = female. Md. = married.
In Diagnosis column the figures, *e.g.* 3/7 means 3 weeks' history from time of onset.
Arteries +, ++, or +++ indicate varying degrees of thickness of the retinal arteries.

CLINICAL NOTE ON ADVANCEMENTS.

BY

ERNEST E. MADDOX, M.D., F.R.C.S.Ed.

BOURNEMOUTH.

WHEN we advance a tendon we are apparently content if it does not slip, and if it affixes itself firmly to the sclera somewhere or everywhere. But is this enough? Might we not have some choice as to the position of the fixation? I think so; and have for some time endeavoured to provoke the tendon's attachment to the original line of insertion. After snipping down the stump with scissors, which lessens the unsightly bulge, I cauterise the line of the original insertion very rapidly, so as not to let the heat have time to penetrate the globe, taking care at the same time not to disturb the endothelium behind the insertion. Indeed, a little animal membrane, or melted hard animal fat, could, if necessary, be placed behind it, to prevent adhesion, but I have not done so yet. Our object should be to retain the normal "arc of contact," so that the tendon can unwind itself from the globe, as much like Nature as possible. The cautery increases the firmness of the adhesion, at the same time.

Although I may not yet have hit upon the best way of doing it, I believe some attempt at reproducing the conditions of Nature should be better than leaving the adhesions to chance. Moreover, in all "tucking" operations for the recti, surface cauterisation of those parts of the tendon about to be brought in contact, will, I believe, be found to give a firmer and more permanent cicatrix than scraping the tendon.

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY.

A meeting of the Society was held on Saturday, October 21st, 1916, at the Royal Infirmary, Manchester, the President, Major A. Hill Griffith, in the chair.

Among the cases shown were the following:—

By **Major Hill Griffith**:—(1) A woman, *æt.* 33 years, with proptosis of the left eye, movements of the eye free in all directions, a distinct new-growth palpable at the back of the eye. Points of interest: there was no perception of light, and the pupil did not react to light, nevertheless the optic disc was of good colour and the retinal vessels were full. (2) A young woman with spring catarrh. She first came for treatment eight years ago. After trying carbon-dioxide snow without effect, radium was applied with excellent results. The patient remained quite well for three years. Two months ago

she came with a slight return of the disease, for which radium is again being applied. The conjunctiva of both eyes now looks quite healthy and normal. (3) A soldier, who had had one eye removed for injury with shrapnel and in the remaining eye showed a glistening piece of metal just below the macula. V. less than 6/60 and J. 16. The eye was quiet and showed no reaction of any kind. The piece had been in the eye for over two months. On the question of treatment, the members present considered it would be best to leave the case alone. (The galvano meter afterwards showed the metal to be non-magnetic.)

By **Dr. Gray Clegg**:—(1) A case of persistent hyaloid artery complete from the disc to the centre of the posterior surface of the lens. The posterior third was very dark, and appeared to contain blood. (2) A case of tuberculous iritis in a boy, *æt.* 16 years. The patient was admitted into hospital in June, 1916, with keratitis punctata and many grey nodules on each iris. No details of the fundus could be made out. R.V.=J.19. L.V.=Hand movements. He was treated with tuberculin emulsion and received nine injections. In four months there was only one nodule on the right iris and none on the left. R.V.=J.16. L.V.=J. 20. (3) A case of tuberculous iritis in a boy, *æt.* 6 years. Two years ago the left iris was covered with numerous tuberculous nodules. Under treatment with Mehnarto's contratoxin the nodules had entirely disappeared. The iris, however, was lustreless, and the pupil closed. The right eye was healthy.

P. J. HAY.

ABSTRACTS.

I.—QUININE AMBLYOPIA.

Weeks, John E.—Report of cases of quinine amblyopia, with remarks. *Trans. Amer. Ophth. Society*, Vol. XIV, Part ii, 1916, page 593.

Weeks, of New York, reports three cases (given in abstract below) of amblyopia following the administration of quinine or of its derivatives.

1. A lady, aged 51 years, received about 315 grains of quinine hydrochloride *per rectum*, and although some of the liquid escaped during the injection, it was thought that about one-half of the quinine was retained. Fourteen and a half hours afterwards, the patient was found to be blind, with dilated pupils. There were complaints of tinnitus and deafness. When examined by Weeks twenty hours after the injection of the quinine, the vision and pupils were as noted.

The optic discs were very pale and the retinal vessels possessed about two-thirds their normal dimensions. The retina was œdematous and pale throughout, except at the macula. The treatment included repeated high, copious, saline enemata; glonoin, 1/100 grain every three hours; strychnine, 1/40 grain, three times a day.

On the following day, the patient could distinguish a bright light; 24 hours later, she could see hand-movements; and on the third day, could count fingers at 4 inches. Seven days after the sight had failed, it was found that the field of the right eye fell everywhere within the 10° circle, except on the temporal side where it extended to about 24°, whereas that of the left eye was confined to the 15° circle. Ten days after the failure of sight, V. 20/30 —., and on the following day the lumen of the retinal arteries was found to have become irregular and the vessel walls thickened. Improvement in these respects followed the administration of caffein, 2 grains, in addition to the glonoin and strychnine. Forty days after the onset of the amblyopia, the light perception, as tested by Snellen's photometric letters, was 75 per cent. About a month later, Parinaud's photometric bars indicated a light perception of 80 per cent. V.=20/20—. The optic discs presented about two-thirds normal capillarity. The retinal arteries had about one-half their natural size, while the veins were but slighly narrowed. The fields for white were still concentrically contracted, but approaching their normal limits. Field for red very small. Reduction in the light sense was believed by Weeks to account for the patient's constant complaint, that she could not see the light, even although vision had become practically normal.

2. A girl of 16 years was treated for pneumonia by the administration in all of 2 grammes of optoquin, spread over 36 hours. Four hours after the last dose, the girl complained that she could not see. Six days later, she began to distinguish light, and in six weeks' time, V.=20/30. Forty-four days after the onset of the mischief, she was first examined by Weeks. V.=20/30. Optic discs paler than normal, but not markedly so. Retinal arteries reduced to about one-half their usual size. Walls of the retinal blood-vessels thickened. Fields: R.E. above, 20°; below, 15°; nasally, 20°; temporally, 35°. L.E. above, 25°; below 25°; nasally, 20°; temporally, 35°. The white fields, estimated about two months after the optoquin had been taken, showed loss of most of the lower halves, more pronounced twelve weeks later, when V.=20/30 —. Colour vision "very indefinite." Light perception about 80 per cent.

3. This is a supplementary report of a case published in 1881 by the late H. Knapp (*Archives of Ophthalmology*, Vol. X, 1881, p. 220). Briefly, loss of vision followed the administration of quinine by the rectum and the mouth. The patient was seen by Knapp almost a

year after these occurrences. V. was 20/20, and colour perception was normal. But the optic discs were white, the retinal arteries very small, and there was a moderate concentric contraction of the visual field in each eye. Two years later, quinine was again given, and again the child developed amblyopia and became green blind. The fields were much contracted. She lost her green blindness, and recovered V. of 20/30. The optic discs, however, remained white, the retinal vessels small, and the fields contracted.

The condition when the patient was examined by Weeks, some thirty-eight years after, was as follows.—R.V. 20/30, L.V. 20/40. Vertical nystagmus. The optic discs were almost chalky-white, and the blood-vessels few in number and reduced in size. The fields of vision showed concentric contraction for white and for colours.

S. S.

II.—OPHTHALMOLOGY AND INTRA-CRANIAL DISORDERS.

(1) **Grey, Ernest G.—Studies on the localization of cerebellar tumours. V. The cranial nerves.** *Bulletin of the Johns Hopkins Hospital*, September, 1916.

(2) **Cooke, Clinton T.—The appearance of the fundus oculi in general paresis, maniac depressive insanity and dementia præcox.** *Ophthalmology*, October, 1916.

(3) **Elsberg, Charles A.—Intracranial surgery and its relation to ophthalmology.** *New York State Journal of Medicine*, November, 1916.

(1) This is the fifth of a series of papers on the localization of tumours in the posterior cranial fossa, and is based on the analysis of sixty-three cases of intra- or extra-cerebellar tumour, confirmed at operation or on *post-mortem* examination. The author deals with lesions affecting all the cranial nerves; in this abstract his observations on the optic and oculo-motor (third, fourth, and sixth) nerves only will be referred to. For the purposes of analysis, subtentorial neoplasms are divided into four groups: (1) Unilateral cerebellar tumours; (2) median cerebellar tumours; (3) cerebellopontine (unilateral recess) tumours; (4) median extra-cerebellar tumours.

Optic Nerves.—Although choked disc has long been recognised as a very usual symptom of cerebellar tumour, cases have been reported (and verified by operation) in which it has been absent throughout. In all but six of Grey's sixty-three cases there was definite choked disc; the fundi in these six cases, with one exception,

showed minor changes, regarded by the author as indicative of increased intracranial pressure. The tumours in all these six cases were intra-cerebellar. While choked disc in itself has no appreciable localizing value, it may have some importance in this respect when associated with other signs. Its early appearance and high degree, when accompanied by some of the so-called cerebellar symptoms, afford valuable confirmatory evidence of a subtentorial localization of the tumour.

Oculo-motor Nerves (third, fourth, sixth).—Weakness of the muscles supplied by the third, fourth, and sixth nerves in cerebellar tumours is rarely due to primary involvement of these nerves or their nuclei, but is usually the result of peripheral involvement, of a secondary nature. It is probable that paralysis of the third and sixth nerves is mainly dependent upon the situation of the new growth below the tentorium and the individual configuration of the lower surface of the brain and the base of the skull. For the purpose of localizing tumours in one or other part of the posterior cranial fossa, paresis or paralysis of these nerves has practically no significance.

Nystagmus and impairment of conjugate deviation, although common, appear to have but little localizing value. While defect or loss of conjugate deviation is generally towards the diseased side, paralysis of such movement towards the opposite side has been observed in one case; in several instances conjugate movements in all directions were restricted. The rule which states that the nystagmus is slower and coarser with the eyes turned towards the tumour is subject to many exceptions. When there is a definite and persistent difference in the size and rate of the jerks, with the eyes in the lateral positions, the nystagmus is usually suggestive of the side of the lesion. J. B. LAWFORD.

(2) **Cooke**, of Seattle, analyses the results of ophthalmoscopic examination in 24 cases of general paralysis, 28 of dementia præcox, and 14 of maniac depressive insanity. He enumerates the changes found, chiefly of a minor kind, *e.g.*, pallor of disc, blurring of disc outline, and vascular changes. Changes in the blood-vessels of the retina were noted in 75, 65, and 78 per cent. of the above-mentioned diseases, and, generally speaking, the similarity of the fundus changes in dementia præcox and maniac depressive insanity to those found in general paralysis seems to indicate an identity of ætiology. A. J. BALLANTYNE.

(3) **Elsberg**, of New York City, discusses some of the ophthalmological problems encountered before, during, and after his last two hundred operations for intracranial disease.

Papillædema and its relief by operation.—We do not know why papillœdema should occur early in some varieties of expanding intracranial disease and be a late comer in others, where intracranial

pressure is just as high. In two of Elsberg's patients with huge tumours of the frontal lobes, and in another with a large tumour on the under surface of the right temporal lobe, repeated examination failed to discover any changes in the fundi. In two cases of large growths in the cerebello-pontine angle there was but slight enlargement of the retinal veins, while a small tumour, the size of a cherry, in the angle, caused a most intense choking of the discs, with hæmorrhages. In addition to increased pressure of fluid in the vaginal sheaths of the optic nerve, there must be some additional factor, as yet unknown. In many instances the amount of internal hydrocephalus is concerned, but, on the other hand, there is the fact that some cases of internal hydrocephalus run their course without any change in the discs, or, if there is marked downward distention of the third ventricle, with primary atrophy.

To show how rapid may be the onset of papillœdema and hæmorrhages, the author quotes the case of a woman, admitted with symptoms of midbrain tumour, but without any changes in the fundi. Two days later, she had a papillœdema of 4 D. in each eye, with hæmorrhages. Again, a patient admitted with the symptoms of a left cerebellar tumour of six months' duration showed a papillœdema of almost 4 D., and on the following morning both fundi showed large hæmorrhages.

Papillœdema may subside with great rapidity. Thus, in one case mentioned by Elsberg, the swelling was reduced from 6 D. to 1 D. twenty-four hours after operation, and in another, a swelling of 2 D. subsided entirely within four hours after operation for meningeal hæmorrhage.

One of the most frequent questions asked prior to an operation for brain tumour is: *Will sight improve or not?* The authors experience leads him to believe that papillœdema of short duration, even when very advanced, may after operation disappear, and normal vision be regained. Sudden blindness from acute choking of the discs is usually recovered from after operation. On the other hand, loss of vision from long standing papillœdema is usually permanent. Some vision has been restored in a few patients who had a marked internal hydrocephalus associated with the new growth. "In most instances if vision is lost for 24 hours from the advanced choked discs, it is usually lost for good."

Patients are often first seen when choking of the discs is already marked. In 63 cases of brain tumour the result of the first examination of the eyes was as under:

The fundus was normal in	24
Papillœdema of 1 D.	6
Papillœdema of 1-2 D.	11
Papillœdema of 3-4 D.	13
Papillœdema of 5-6 D.	9

Only 28 of the above patients complained of failing vision, but 39 had more or less well-marked swelling of the discs, and 22 had a papillœdema of a high grade. That 62 per cent. of the patients should have sought aid only when they had marked papillœdema, and 35 per cent. only after the swelling of the discs was far advanced indicates that in many cases ophthalmoscopic examinations are put off too long. During the past year Elsberg has twice operated upon a patient with marked papillœdema, in whom a very successful operation, with rapid subsidence of the swelling of the discs, failed to save eyesight.

As regards the vexed question, whether there is any relation between the side of the tumour and the papillœdema, the author, in looking over the records of one hundred patients, has found that in fewer than 40 per cent. was the swelling most marked on the side of the neoplasm. In about 60 per cent. of patients with a new growth in one or other frontal lobe, the papillœdema was most advanced on the side of the tumour. A study of the post-operative subsidence of the papillœdema brought out certain interesting facts. In 84 per cent. of the patients the papillœdema of the eye on the same side as the tumour subsided much more rapidly after the growth was removed than that of the opposite side. If a decompression was done, the papillœdema subsided more rapidly on the side opposite to that of the tumour, unless the latter was located on the same side as the decompressive operation. If a bilateral decompression was performed, then the eye contralateral to the side of the tumour improved more rapidly than its fellow.

The effects of direct pressure upon the nerves and the chiasm and its relief by operation.—Elsberg's experience leads him to agree with Foster Kennedy in claiming that enlargement of the blind spot is especially frequent in tumours of the frontal lobes, and that in a suspected frontal new growth a paracentral scotoma, due to blocking of the more sensitive macular fibres, is of frequent occurrence. His experiences of hypophyseal diseases accord with those of Cushing and Walker. He has met with homonymous hemianopsia, even quadrant defects, in a number of patients. If the fields are tested with small test objects, small quadrant defects in the temporal fields for form, and especially for colour, will frequently be discovered.

It is well-known that patients suffering from hypophyseal disease are often blind of one eye, while the other is still normal or has a well-marked defect in the temporal field. In one such patient Elsberg exposed the sella Turcica, and found that an hypophyseal adenoma had surrounded the chiasm and had also grown around the left optic nerve.

Can sight be regained in an eye blinded from direct pressure on the chiasm, or the optic nerve, when the pressure is removed ?—A

return of vision is possible as long as the pupil will contract to light. In one of Elsberg's patients with an hypophyseal cyst, with one eye blind for over six months and the other hemiopic on the temporal side, a sellar decompression and evacuation of the cyst were followed not only by great improvement of the hemiopic field, but also the return of some sight in the blind eye. The following case is also of interest in this connection.—A patient, who had been acromegalic for years, after a febrile affection, developed signs of an infection of the sphenoid sinuses, with basilar meningitis and rapid deterioration of vision. Within two weeks the right eye was blind, and the left eye showed a large temporal defect. As the signs of meningitis disappeared after drainage of the sphenoid sinus, sight rapidly returned in the blind eye and the hemiopic field became rapidly enlarged. Both fields were normal two weeks after improvement had begun. S. S.

III.—AFFECTIONS OF THE EYE IN DYSENTERY.

(1) **Cosse and Delord.—Some cases of conjunctivitis met with in dysenterics. (Quelques cas de conjonctivite observés chez des dysentériques.)** *Annales d'Oculistique*, janvier, 1917.

(2) **Morax, V.—Iritis in the course of a dysentery-like intestinal infection. (Iritis au cours d'une infection intestinale dysentériforme.)** *Annales d'Oculistique*, janvier, 1917.

The following abstracts refer to affections of the eye in dysentery or in dysentery-like conditions. A case appears to have been made out for the occurrence of conjunctivitis and iritis as metastatic complications of these affections.

(1) **Cosse** and **Delord** observed a certain number of cases of conjunctivitis during the course of an epidemic of bacillary dysentery at Chartres in the summer of 1916. They find no reference to this complication in the literature they have consulted.

The cases are grouped under two heads: 1. those accompanied by affections of the joints; and 2. those not so accompanied.

1. The first group included eight cases. The symptoms of dysentery were more serious, as a rule, than those of the second group. The conjunctivitis, which lasted for eight or ten days, made its appearance about the fifteenth day of the disease. At this time the articular complications (pain, swelling, and effusion) were observed, and affected chiefly the knees or the tibio-tarsal joints, although the shoulder or the elbow was sometimes involved. The articular lesions lasted for a considerable time, and confined the

patient to his bed or to his room for several weeks. On occasion, the "rheumatism" preceded the conjunctivitis or accompanied the last-named. Sometimes the enteritis was cured when the conjunctival or articular manifestations declared themselves. In one paradoxical case (Obs. XII) conjunctivitis preceded both the diarrhœa and the affection of the joints. A non-specific bacillus was found in the stools by G. Rosenthal. As to the characters of the conjunctivitis, both eyes were affected at the outset, inflammatory signs were limited to the palpebral conjunctiva, especially to that of the lower lids, and the eyelids were agglutinated after sleep. No follicles or granulations. Glands not involved. Bacteriological examination of the moderate amount of conjunctival secretion, by means of "smears" and cultures, negative. Some cases were treated with argyrol, but more were left alone, and cure was complete in ten days. In one case alone were peripheral keratitis and iritis observed as complications of the conjunctival affection.

2. The second group included four examples of a form of mild conjunctivitis, the stress of which fell almost wholly upon the lower palpebral conjunctiva. It affected both eyes, and made its appearance five to twelve days after the beginning of the dysenteric symptoms. It was associated with a minimal amount of secretion from the conjunctiva. The condition underwent spontaneous cure in six or seven days, or in even a shorter time.

In discussing the conditions described above the authors place wholly aside any question of direct infection, as from fingers or linen. They conclude that the conjunctivitis was of toxic origin and metastatic in character, directly dependent upon the dysentery. They question the propriety of speaking of it as an "inflammation," which implies an infection, and would rather look upon it as a conjunctival manifestation of toxic origin.

Appended to Cosse and Delord's article is a brief account of two cases, where the appearance of articular rheumatism was preceded by mild but definite conjunctivitis. S. S.

(2) The patient whose case is described by **Morax**, of Paris, was amongst the sufferers in an epidemic, resembling dysentery. This outbreak, which affected nearly 250 men, occurred in barracks. It was characterised by acute enteritis, with frequent and liquid stools, tenesmus, and rise of temperature, and the common association was with articular manifestations. No precise conclusion could be drawn from such bacteriological examinations as were carried out, although the latter, at least, went to exclude amoebic dysentery. Serum treatment met with no success in the several cases where it was tried.

Morax's patient had been affected for about a fortnight when his eyes were noticed to be injected, and to have a little secretion at the inner canthus. About a week later, lively pain was experienced in

the right foot, to be followed by a similar occurrence in the left foot, the knees, the left elbow, and the sternum being affected a few days later. There was renewed conjunctival mischief involving both eyes. Thirty-five days after the dysentery began, sight became cloudy, and more violent pain set in as regards the right eye, but did not last long. After dilatation of the pupil with atropine, a few small deposits of pigment were found upon the anterior capsule of the lens of the affected eye. A slight relapse of the iritis made its appearance ten days later, to be followed by a third relapse a little later. It is to be noted that the temperature-curve had shown fluctuations at about the periods of the relapses of the iritis.

Morax briefly mentions the case of an officer from Salonika, who had a unilateral and somewhat violent attack of iritis, succeeding enteritis of dysenteric type. He made a good recovery. He also alludes to a case by Vossius (*Die ophtalmologische Klinik*, 1904, S. 17) of a patient, aged 28 years, who developed in the course of the third week of a typical attack of dysentery, prostatic pain, and a muco-purulent urethral discharge, free from gonococci. Shortly after, he developed conjunctivitis of both eyes (no gonococci), followed by poor sight, and kerato-irido-cyclitis. Normal vision was eventually regained. The dysenteric nature of the general affection was established by the serum reaction; the patient's serum, diluted to 1/320 and to 1/1000, strongly agglutinated Shiga's bacillus.

Following a suggestion recently made by N. Fiessinger and E. Leroy (*Soc. méd. des Hôpitaux*, 8 décembre, 1916), Morax is inclined to regard the articulo-ocular syndrome of dysentery as analogous to the blennorrhagic syndrome. S. S.

IV.—HÆMORRHAGIC RETINITIS.

Amsler, Marc.—Contribution to the study of hæmorrhagic retinitis, and especially of its ætiology and prognosis. (Contribution à l'étude de la rétinite hémorragique, notamment de son étiologie et de son pronostic.) *La Clinique Ophtalmologique*, mai, 1916 (Abstract from a *brochure* published at Lausanne by *Art et Science*, price 2fr. 50).

We may transcribe the author's conclusions as given in this abstract. The original deals, we are told, with the case-histories of 164 cases occurring in 40,000 patients attending the private and public clinics of Professor Eperon. In using the term "hæmorrhagic retinitis," the author explains that he chose it for convenience, without prejudice to the question of actual inflammation of the retina.

Hæmorrhagic retinitis occurs in 4 per 1,000 cases, and has various ophthalmoscopic appearances. It occurs especially between the years 50 and 70, and more often in women than in men.

It is usually unilateral and seriously affects vision.

It is almost always a manifestation of general disease.

In more than one-half of the cases arterio-sclerosis is the cause. Among the ætiological factors of the remainder are to be found cardiac diseases, albuminuria and glycosuria, arthritism and obesity, the menopause, and, more rarely, influenza.

Not infrequently, the hæmorrhage may be caused by some effort.

The prognosis of hæmorrhagic retinitis is good in 40 per cent. of cases. The favourable factors in these are, the female sex (especially at the menopause between 45 and 60 years); when the hæmorrhages are pre-retinal, that is, hæmorrhagic retinitis in the strict sense, the hæmorrhages being few and peripapillary; a good condition of health, especially as regards the digestive system; appropriate treatment.

The prognosis is bad in 60 per cent. of cases. The following are among the unfavourable conditions, namely, the male sex, advanced age, early arterio-sclerosis, fatigue, overwork, and general weakness.

The appearance of hæmorrhagic retinitis in an arterio-sclerotic subject makes one anticipate cerebral apoplexy. Thrombosis of the central vein is less rare than has been stated (1 in 2,000). We believe that its prognosis is not invariably bad.

ERNEST THOMSON.

V.—AFFECTIONS OF THE DUCTLESS GLANDS.

(1) **Lamb, R. S.—The internal secretory system in ophthalmology with special reference to goitre.** *Annals of Ophthalmology*, April, 1916.

(2) **Schirmer, Otto.—Internal secretions and eye diseases.** *New York State Journal of Medicine*, January, 1917.

(1) **Lamb**, of Washington, first reviews the literature of his subject, and then states the purpose of his paper, which is: (1) to bring to the attention of ophthalmologists the signs which have been accepted as indicative of certain perversions of the internal secretory system (with special reference to goitre), and (2) at the same time to offer suggestions regarding them, in order to facilitate the identification of these cases, and thereby to obtain an early and

correct diagnosis of the condition. He believes that thus much suffering may be saved, and chronic invalidism avoided. A factor which complicates all such calculations is the extraordinary interplay in the body between the various glands; an influence which activates one gland may, through an excess of its secretion, stimulate quite another organ to a great increase of activity. The various signs on which Peter lays stress are described each in turn. A number of cases are quoted, and the pathological causes underlying the morbid phenomena are discussed. Finally, the various cases met with are classified, with a view of simplifying the task of those who would wish to follow in Lamb's footsteps.

There are doubtless features in the paper which will arouse the critical faculty of a good many readers; but it is painstaking and suggestive, and is well worth a careful perusal. R. H. ELLIOT.

(2) **Schirmer**, of New York City, discusses broadly the symptoms and bearings of disordered functions of the so-called "ductless glands." He then speaks of the eye symptoms that may accompany those conditions.

Pressure affections of the optic nerve accompanying disorders of the hypophysis come in for particular notice, and the author lays stress upon the fact that enlargement of the gland may go with over-function, as well as with under-function. Although temporal hemianopsia is the common type of disturbance, there are cases where the visual fields show concentric contraction or homonymous hemianopsia in affections of the hypophysis. He comments upon the powers of resistance often manifested by the fibres of the optic nerve. In other words, pressure may abolish vision without destroying the life of the fibre, as in cases where by surgical or other means pressure has been relieved and sight more or less completely restored. He mentions the case of passing temporal hemianopsia in multiparæ during the last period of pregnancy (A. v. Reuss). Under such circumstances the hypophysis swells considerably during pregnancy and shrinks as soon as the child is delivered. With every subsequent pregnancy, the gland becomes larger, so that in some cases pressure on the chiasm may occur. It yet remains to be proved that changes in the hypophyseal secretion can produce eye symptoms.

The ocular complications in disturbances of the pineal gland include choked disc, optic atrophy, and anomalies of the eye muscles.

Schirmer thinks it probable that all the eye symptoms of Graves' disease are a direct consequence of the over-function of the thyroid gland. He explains the exophthalmus by an early swelling or even hypertrophy of the fat tissue, which would account for the fact that the symptom often persists after death or in cases otherwise completely cured by surgical intervention. According to Kocher, exophthalmus

persists in 45 per cent. of the cured cases. The affections of the optic nerve (atrophy and inflammation) in Graves' disease are regarded by Schirmer as the direct result of the toxic action of thyroidin, inasmuch as such are known to have followed the experimental and medicinal administration of that drug.

As to affections of the eye in tetany, the author puts on one side the single case of optic atrophy reported by Hanke, since the optic nerve has always been found to be healthy in the innumerable instances where the disease has been experimentally induced in different animals. The eye muscles, however, sometimes, though rarely, participate in the tetanic spasm—blepharospasm, diplopia, and contraction of the pupil have been observed. Conjunctivitis has followed experimental extirpation of the parathyroids, and has been noted in tetany itself (Falta and Kahn). Schirmer looks upon the associated cataract as due to changes in the epithelium of the ciliary body; and Peters, indeed, has found microscopical evidence of such in the ciliary epithelium. In children, the cataract of tetany mostly affects the lamellar type, but in adults it is usually of the cortical type, and may mature rapidly.

Schirmer concludes a suggestive communication by alluding to Abderhalden's method, and outlining some of the work that has been done on those lines, more especially by v. Hippel, to ascertain the condition of the ductless glands in diseases of the eye.

Keratoconus is the one affection of the eye in which the Abderhalden method has given positive results in the great majority of instances. Years ago, it will be recalled, Siegrist claimed that disease as a malady due to under-function of the thyroid gland. v. Hippel's results point in a similar direction. They indicate that in keratoconus the endocrine glands are in a pathological condition. The clinical examination was negative, and especially sexual anomalies were always absent.

v. Hippel also often obtained positive results from the Abderhalden reaction in glaucoma.

Römer and Gebb claim to have found by the Abderhalden method that the biological relation of the lens albumen toward blood-serum is different in cataractous and non-cataractous patients. Abderhalden and v. Hippel have repeated these experiments and have failed entirely to find these differences. Schirmer concludes, therefore, that no new light has so far been thrown upon the ætiology of cataract.

The author ends by suggesting that the study of anomalies of internal secretion will lead to valuable results, not only in the hands of the general physician, but also in those of the ophthalmic surgeon.

Discussion.

Henry H. Tyson, New York City, had reported in 1910 a case of Graves' disease with optic neuritis followed by optic atrophy.

As to the suggested association between under-function of the thyroid and keratoconus, he had found the opposite condition, namely, over-function, in one case recently, but the keratoconus had antedated the other condition by about six months. He drew attention to a sign that could be elicited early in many cases of over-function of the thyroid gland, namely, a widening of the palpebral fissure upon forced fixation of an object, held midway between and upon the same plane as the eyes, and close to the latter.

Edgar S. Thomson, New York City, regarded the exophthalmus of Graves' disease as due to a low grade inflammatory condition of the tissues. In a man of about 37 years, who had been treated surgically for Graves' disease, with marked exophthalmus, operation was followed by exophthalmus and by a gradual œdema of the ocular conjunctiva of both eyes, so that it eventually became impossible to close the eyelids. Removal of tissue became necessary, and microscopical examination showed an extensive connective tissue hyperplasia, which had gone on to organisation. The exophthalmus remained, and the speaker surmised that a similar tissue change had taken place in the posterior part of the orbit, thus accounting for the protrusion of the eyes. S. S.

VI.—MYOPIA.

(1) **Koster, W.—On school myopia: its cause and treatment. (Over schoolmyopie haar ontstaan en behandeling.)** *Nederlandsch Tijdschrift voor Geneeskunde*, 1916, Part I.

(2) **Pollock, W. B. Inglis.—The reduction of myopia in children of school age.** *Glasgow Medical Journal*, October, 1916.

(3) **Sidler-Huguenin.—Can the development of myopia be arrested and its degenerative changes be prevented?** *Archives of Ophthalmology*, Vol. XLV, No. 6, November, 1916.

(1) School myopia and progressive malignant myopia are two quite different diseases. Some authors have tried to prove their identity statistically. School myopia is caused by over-exertion and heredity. In **Koster's** opinion, it is not the direct action of accommodation or convergence that has a bad influence, but these eyes are suffering from a very chronic, slight chorio-retinitis on an hereditary basis. By the effort of accommodation the eyes, especially the posterior parts, are made hyperæmic, and under the combined influence of these two factors, the posterior part of the

globe becomes ectatic. A logical treatment of school myopia, as considered from this point of view, is (1) against the chronic retino-choroiditis small doses of iodine and salicylates, continued for a long period, avoiding of congestion to the head, and application of radium as far back on the globe as possible; and (2) no over-exertion by reading and writing without glasses, in suitable cases, even with *plus* glasses. G. F. ROCHAT.

(2) The article by **Pollock**, of Glasgow, on the reduction of myopia in children of school age, is of interest. One must hope that other ophthalmologists will have equally good results. The treatment is that by prolonged use of atropine, associated with removal from school, or, at any rate, with complete cessation of near work. Pollock is fortunate in having to deal with the first School Board in Scotland to introduce the myopia class, namely, the School Board of Govan. The author says that his work in connection with the treatment of myopia began about six years ago with the extension of the use of atropine for testing the refraction. "It is," he says, "common knowledge that after a week of atropine the myopia is about a dioptre less than prior to using the atropine. This change has always been regarded as due to the relaxation of the accommodation, and accordingly no further reduction in the myopia was thought to be possible by continuing the mydriasis. My reason in the first instance for an extended use of atropine was to prevent the patient from doing near work, and thus to give the eyes a more thorough rest; and also, if possible, to prevent a further increase in the myopia. I found, however, that in certain cases after two or three months of treatment the myopia not only stopped increasing, but actually showed a decrease. This induced me to persevere for much more prolonged periods, and the result has been most satisfactory."

It is, of course, true that a great many ophthalmic surgeons must have employed fairly prolonged atropinization in cases of myopia, but, so far as the reviewer is aware, no one has ever published a series of cases showing a reduction of myopia following the use of this or of any other drug, in the least comparable to this series. The cases are four in number. The details follow:—

(1) A girl, aged 7 years, showed by retinoscopy after a week of atropine 11 and 9 D., in the vertical and horizontal meridians respectively, in each eye. After two months the retinoscopy was right eye 8 and 6 D., left eye 9 and 7 D. One month later, each eye was 7·5 and 6 D. Six months later, the right eye was 6 and 4·5 D., the left 6 and 4 D. Two months later, each eye had 6·5 and 4·5 D. This was the last measurement, on 28th October, 1914. Reduction 4·5 D. in ten months.

(2) A girl, aged 9 years, showed, after a week of atropine, 18 and 16 D. in each eye. Seven months later, the right eye had 14 and

10 D., the left 13 and 9 D. Last examination September, 1914. Reduction 4 D. and 5 D. in R.E. and L.E.

(3) A boy, aged 11 years, after a week of atropine, showed 11 and 6 D. in the right eye, and 7 and 2 D. in the left. Three months later, the refraction was 8 and 4 D. in the right, and 6 and 1 D. in the left. Last examination May, 1914. Reduction 3 D. and 1 D. in R.E. and L.E. respectively, in three months.

(4) A girl, aged 9 years, presented, after a week of atropine, 13 and 12 D. in the right eye and 12 and 11 D. in the left. In three months, the right eye gave 10 and 8 D., the left 9 and 8 D. Last examination February, 1915. Reduction 3 D. in each eye in three months.

Pollock states that he has also obtained a reduction of 3 or 4 D. in cases of low myopia and that some of these have become converted into low hypermetropia. Relapses have occurred in some cases during the summer months when the school eye clinics are closed, and treatment was not carried out.

These are the main facts stated by the author, but he also refers to details of constitutional treatment, to atropine poisoning and atropine dermatitis, and to the well-known difficulty in getting near work really suppressed. The recently instituted myope class has, however, justified all his expectations. ERNEST THOMSON.

(3) **Sidler-Huguenin**, of Zurich, propounds the question whether the development of myopia can be arrested and its degenerative changes prevented. As his answer is an unqualified negative, and as it is based on very painstaking work, it cannot be lightly brushed aside, despite the fact that his conclusions are little short of revolutionary.

By the aid of carefully adjusted radiographs, he has satisfied himself that there is no close relationship between the contents of the orbit on one side and the refraction of the eye on the same side; he consequently rejects Stilling's theory. He next considers a series of 150 anisometropes, each of whom practically used only one eye. These patients gradually became more and more myopic in the worse eye, which he states they did not use. From this he deduces that "the use of the eye has nothing to do with the degree or the deleterious consequences of a near-sighted eye." He quotes Steiger in support of his position. Is it a valid argument that because an eye becomes more myopic when its fellow is working, it would equally become more myopic, if both eyes were laid aside from work? In other words, does an eye ever *really rest* whilst its fellow is working? The answer is almost certainly in the negative to both questions. In the 150 cases quoted there was evidence of heredity of myopia in 18 only. Yet the author says that "we must consider heredity as the most frequent ætiological factor in the development of myopia."

In 4,000 myopic patients the author found disease of the macula in 218 eyes, and of these 49 had a myopia of less than 10 D. This corresponds with Schweizer's results in Haab's clinic. There, in 5,000 myopic eyes, 265 showed macular disease, and in 44 of them the myopia was under 10 D. In the 150 anisometropic cases already quoted, corneal changes were present in 78 eyes, whilst in the 4,000 myopes corneal maculæ were present in 187. The writer is inclined to agree with Meyerhof and others, that the corneal opacity is the cause of the development of the myopia, or, at least, that there is an association between opacity of the cornea or lens and myopia. He founds this view largely on the fact that in his series of cases a hereditary history of myopia is present in only one of the 78 eyes which showed corneal maculæ. He confesses himself at a loss to explain the causative relationship which he accepts. He suggests that in highly anisometropic eyes, the retina and choroid degenerate rapidly on account of lack of function, but his arguments fail to carry conviction,

He next has something trenchant to say of the bad results which follow the removal of the lens for the relief of high myopia. Again, his view that the use of the myopic eye is probably more favourable than non-use, is based on arguments that will provoke a challenge in a good many minds.

Some stress is laid on the opinions expressed by intelligent patients that a general treatment of their myopia had not affected the course of the eye condition. It may, however, be questioned whether such evidence is of any value whatever. When the writer goes on to say that the usual directions which we give these myopic patients have but little effect, and, again, that myopia gradually increases, irrespective of external influences, to a degree which is dependent upon hereditary factors, one cannot but feel that such fatalism is in excess of what the arguments justify, and that to follow him in practice would be to set back the clock of progress.

On the strength of some very interesting figures, the author has come to the conclusion that "myopia may remain stationary just as well with under-correction as with full-correction," and again, "that full correction is not always capable of preventing the myopia from progressing."

It must be admitted that the writer has had a large clinical material, and has made much use of it, but the pessimism which he manifests towards one and all of the accepted means for the control of myopia will fail to appeal to a great many surgeons. To say that myopia is "a congenital evil which can only be influenced by the proper selection of individuals for marriage," is, from the point of view of practical results, a mere waste of breath. The article is interesting and stimulating; it is the expression of the thoughts of a man who dares to leave the beaten path, and any criticism should

therefore be offered in a spirit of reluctance and respect. It is certainly a paper which every thinking ophthalmologist should read.

R. H. ELLIOT.

THE ÆTIOLOGY OF IRIDO-CYCLITIS.

(1) **Keppeler, Eugen.—Gonorrhœal iritis.** *Klin. Monatsbl. f. Augenheilkunde*, February-March, 1915.

(2) **Dernehl, P. H.—Chronic prostatitis a probable factor in iritis.** *Ophthalmology*, April, 1915.

(3) **Reber, Wendell.—Iritis due to influenza.** *Ophthalmic Record*, November, 1915.

(4) **de Schweinitz, G. E.—Concerning focal infections in their relation to certain disorders of the uveal tract.** *Ophthalmic Record*, December, 1915.

(5) **Reber, Wendell.—Concerning the etiology of iritis as determined by laboratory methods, and its treatment, especially by bacterins.** *Ophthalmic Record*, May, 1916.

(6) **Brown, E. V. L. and Irons, Ernest E.—The etiology of iritis.** *Transactions American Ophthalmological Society*, Vol. XIV, Part ii, 1916, p. 495.

(7) **Francis, Lee Masten. — Recent ideas concerning the diagnosis and vaccine therapy of irido-cyclitis. Report of a case.** *New York State Journal of Medicine*, September, 1916.

(1) Reviewing the literature of iritis gonorrhoica from the earliest period, **Keppeler**, of Tübingen, indicates that modern clinical descriptions of the disease have little to add to that given by Mackenzie, of Glasgow (1854). From Mckenzie downwards, most writers agree that gonorrhœal iritis is a disease of acute onset, of great severity (pain, redness, chemois, etc.), with deposits on the cornea and iris, and in many cases (Sidler-Huguenin) a lens-like mass of fibrinous exudate in the anterior chamber. In three of Sidler's cases gonococci were found in the blood, and in one in the aqueous. Other characteristics of the disease are its good and rapid response to treatment, coupled with a relapsing tendency. It affects exclusively males between the ages of 20 and 45. It is unilateral. Usually other evidences of systemic infection are present, in the shape of articular pains, etc., but these may be absent. That the other parts of the uvea are involved is often proved by the discovery of vitreous opacities after the iritis has subsided. The disease seldom occurs during the existence of an acute gonorrhœal urethritis. It usually appears after the onset of joint affections has indicated the diffusion of the disease. Recurrences are usually preceded by increase of urethral discharge due to exacerbation of a chronic urethritis or by

an outbreak of articular inflammation. The course is not always favourable. Seclusio and occlusio pupillæ, cataract, and secondary glaucoma may occur.

Local treatment follows the usual lines, and internally we may use quinine, iodides, and calomel. Keppeler specially recommends diaphoresis by means of the salicylates, baths, etc. Other treatment includes the use of gonococcal vaccine, antigonorrhœal serum, and paraspecific serum therapy. A. J. BALLANTYNE.

(2) **Dernehl,** of Milwaukee, reports three cases of males with recurrent iritis, in whom there was a history of gonorrhœal infection. The periods of infection were respectively six, ten, and twenty years before the onset of the iritis. The prostatic secretion was found to contain pus cells and Gram-negative diplococci morphologically similar to the gonococcus but extra-cellular. He inclines to the view that the chronic prostatitis was the cause of the iritis, and that it was gonorrhœal in origin. A. J. BALLANTYNE.

(3) **Reber,** of Philadelphia, reports a case of iritis which was shown to be of influenzal origin by the complement-fixation test, and the rapid relief of symptoms after the use of an injection of influenzal mixed sero-bacterin (sensitised), after failure with salicylates and the vigorous local use of atropine. When the sero-bacterin was discontinued, and the patient placed on mercury and iodide, a violent relapse of the iritis took place. This again gave way promptly to a double dose of sensitised influenzal sero-bacterin.

J. JAMESON EVANS.

(4) **de Schweinitz,** of Philadelphia, reviews the subject of infective or toxic uveitis, the broad principles of which were laid down by Brailey and Stephenson some twenty years ago in Norris and Oliver's *System of Diseases of the Eye.* In connection with oral sepsis, he directs attention to the work of Smith and Barrett, of the University of Pennsylvania, on the relation of parasitic amœba to pyorrhea alveolaris and the production of toxins by their bacteriolytic action. But it is well to remember that "general oral sepsis or extensive pyorrhea alveolaris need not necessarily be present as the instigator of uveitis. The infection may come on from a small tooth-root abscess or from a blind abscess, the presence of which is unsuspected and often undetected until the X-rays are employed."

Other sources of infection referred to are nose, nasal accessory sinuses, tonsils, skin, uterine cavity, prostate, seminal vesicles, and gastro-intestinal tract.

The sources of infection may come from various regions in one and the same subject, and therefore the discovery of a single focal infection should not check the search for septic areas elsewhere in the body. J. JAMESON EVANS.

(5) **Reber,** of Philadelphia, confesses to a feeling of disappoint-

ment with current statements as to the ætiology of iritis. The present purely clinical classification into syphilitic, rheumatic, gonorrhœal iritis, and so forth, is little in advance of that adopted half a century ago. Not only is there confusion as to ætiology, but confusion also prevails as to the relative frequency of each supposed cause. For example, syphilis as the predisposing factor of iritis has been estimated at from as little as 20 per cent. to as much as 70 per cent., and the same is true of the other assumed causes.

Reber believes that with the modern laboratory methods at our disposal, we should be able to get closer to the truth. In determining this point, the history and clinical aspect of a given case must never be neglected; but neither must a so-called complement fixation test or more than one, if necessary. These reactions may be obtained as regards nine micro-organisms, of which five have been shown to bear a causative relationship to inflammation of the iris, namely, (1) the spirochæta pallida; (2) the gonococcus; (3) the pneumococcus; (4) the streptococcus; and (5) the post-influenzal group.

During the last year, Reber has investigated 15 cases of acute primary iritis, making in every one a complement fixation test for the following: Wassermann, gonococcus, pneumococcus, streptococcus, staphylococcus pyogenes albus and aureus, bacillus coli, and bacillus influenzæ. Of the 15 cases, 5 were syphilitic, both experimentally and clinically; 3 gave a positive gonococcus reaction, although there was no clinical evidence of gonorrhœa; and 1 was positive to the influenza bacillus. In 6 cases no reaction was obtained, but there were other reasons for thinking that of these negative cases 2 were due to syphilis, 1 to tuberculosis, and 1 to gastro-intestinal toxæmia. Briefly, then, of the series of 15 cases, 33 per cent. were certainly syphilitic and 14 per cent. possibly syphilitic; 20 per cent. were gonococcal; 6·5 per cent. were influenzal; 6·5 per cent. were tuberculous; 6·5 were autotoxic; and, finally, 14 per cent. were of obscure origin. Rheumatic iritis does not figure in the list, and, in Reber's opinion, is destined ultimately to disappear from our text-books and literature.

In conclusion, Reber has something to say about treatment. As regards the syphilitic cases, although he admits the efficacy of salvarsan and neo-salvarsan, yet he has unwavering faith in mercury. If a positive gonococcus fixation test be obtained, the indication for bacterin or vaccine treatment is plain, and if there be arthritic involvement, even in the presence of a negative laboratory examination, Reber recommends the injection of Neisser mixed bacterin. If no cause can be determined, the author feels that the injection of influenza mixed bacterin is justified. S. S.

(6) **Brown** and **Irons**, of Chicago, subjected 100 patients affected with non-traumatic iritis to a most complete examination, physical,

laboratory, radiographic, etc., in order to determine the cause of the eye mischief. Attention was also paid to the effects of treatment, as indicative of cause. In the course of their investigations they were struck by the large number of cases in which two or more competent causes of iritis were combined in one and the same patient, and they point out the difficulty under these circumstances of deciding the cause of the disease, especially in hospital patients, in whom multiple infections are frequently found.

The causes of iritis, as enumerated by the authors, may be taken up one by one, as follows:—

1. *Syphilis.*—In 39 cases syphilitic infection had occurred either recently or years before. In 20 of these patients the iritis was obviously syphilitic. Among that number, 13 manifested other clinical evidence of active syphilis. In the remaining 7 the Wassermann reaction was positive in all. Of the 39, 3 other cases were probably syphilitic, to judge from the fact that prompt relief followed the exhibition of antiluetic treatment, although other possible causes of iritis were also present. Syphilis as a cause of iritis was commoner (32·07 per cent.) in the dispensary patients than in the private group (12·76 per cent.). Finally it may be added that the Wassermann test was applied in 98 of the total 100 patients dealt with in the communication.

2. *Dental Infection.*—In 41 patients dental infections, demonstrated by radiographs and confirmed by dental surgeons, were found, and in 18 instances this was recognised to be the cause of the iritis.

3. *Tonsillar infection.*—Under this heading the authors included deep infections of the tonsils, intra-tonsillar and retro-tonsillar abscesses. On the other hand, slight lesions, such as small crypts containing "cheesy" material, unless accompanied by deeper infection, were not counted. In 16 of the cases of iritis the tonsil was clearly the source of infection. Tonsillar infection was found in several cases of iritis due to other causes.

4. *Gonococcal infection.*—This was considered to be the cause of iritis in 9 patients, all males. In all, save one, arthritis was present. A positive gonococcal complement fixation test was obtained in 6 of the 7 cases where it was made. In 7 of the 9 cases the iritis was recurrent, and often associated with recurrent arthritis following exacerbations of urethritis. In 7 of the 9 patients infections other than gonococcal were not found. In the other 2 (both recurrent), tonsillar or dental infections were present, but the recurrences were closely associated with exacerbations of urethritis and arthritis.

5. *Tuberculosis.*—In 8 patients the iritis appeared to be of tuberculous origin. In 7 the evidence was clear, and in 1 presumptive. In 2 of the 7, active clinical tuberculosis was present, and in 5 positive tuberculin reactions, along with other evidences of tuberculosis, were demonstrated by physical examination and

radiographs. In 2 cases focal reactions in the eye repeatedly occurred after tuberculin tests. In none of the 7 patients was there any other discoverable focus of infection.

6. *Sinus infection.*—The authors' experience leads them to believe that there is more difficulty in determining the presence or absence of infection in the sinuses than in any other part of the body. In three of the patients the iritis was considered as due to sinus infection.

7. *Other infections.*—In 3 cases the iritis was associated with non-venereal infections of the genito-urinary tract, including 2 of the prostate.

8. *Combined infections.*—In 17 patients there were several possible causes of the inflammation of the iris, and the authors were unable to determine which infection was responsible.

In 1 case no adequate cause for the iritis could be found.

In conclusion, Brown and Irons offer some interesting speculations with regard to the mechanism whereby iritis is brought about. Despite the commonness of focal infections, iritis and other metastatic lesions are rare. It can be produced in animals by the intra-venous injection of bacteria (Stock), which reach the eye by the circulating blood, and which can be recovered from the inflamed organ. The authors have set up iritis in rabbits by the injection into the venous system of streptococcus, B. pyocyaneus, B. mucosus capsulatus, and gonococcus. But large doses must usually be given, so that a condition of sepsis is created, and even then iritis is by no means always produced. By injuring one eye of a series of rabbits by the intra-ocular injection of minute quantities of dead bacteria, and then when the eyes have healed, injecting living staphylococci or streptococci into the veins, they have found that the injured eyes are more liable to infection than the control eyes. Another point to be considered are changes in the infective organisms themselves. In a streptococcal inflammation of the tear-sac, which had presumably caused iritis, the authors were able to produce iritis in rabbits by the injection of the organisms first isolated from the patient. Later cultures, however, yielded negative results.

Finally, we can scarcely do better than quote the authors' closing words:—"It would appear that not one, but several factors are concerned in the production of iritis, among which may be noted the presence of a focus of an infection in which bacteria may live and multiply, partially protected from the living forces of the host; the periodic entrance into the blood-stream of these organisms and their lodgment in the eye, either by reason of their great number, as in sepsis, or by fortuitous embolism; changes in the eye itself, either in vascular anastomoses or in susceptibility of the ocular tissues, owing to previous injury; and, finally, change in the organism

itself, whereby it may become better able for a time to maintain itself in the tissues of the eye. Such adaptations on the part of the organism do not necessarily imply any demonstrable change in its morphology or cultural reactions." S. S.

(7) **Francis,** of Buffalo, speaking of the recent articles upon the ætiology of irido-cyclitis by Wendell Reber: Brown, Irons, and Nadler; and Brown and Irons, claims that they "blaze a trail in a field which needs exploration, and foreshadow a refinement of diagnosis and therapy which augurs much for ophthalmology." The communications in question have stimulated the appearance of the present contribution.

In searching for the cause of affections of the uveal tract, Francis states that the investigations naturally fall into two groups: 1. the search for a focus from which infection could be transferred to the eye by embolism; and 2. the search for a localized infection in the eye as a part of a generalized systemic process, as syphilis, gonorrhœa, tubercle, etc. Treatment will depend upon which group the condition belongs to. For example, in the first it largely resolves itself into removal of the infective source, as by the extraction of diseased teeth or the enucleation of affected tonsils. In the second group, the co-operation of a laboratory equipped to carry out complement fixation work is essential. The next step is the selection of an appropriate vaccine, which should be autogenous, whenever possible. Failing that, a polyvalent vaccine should be employed. The actual administration of the vaccine should be left to a serologist.

In reference to the value of vaccines, in post-operative infection, Francis relates one very successful case. The patient appears to have developed tonsillitis on the second day after extraction of immature cataract, and coincidently the anterior chamber was partly filled with blood, the iris was muddy, and the pupillary area was "murky." The corneal incision was united, and entirely clear. Ordinary treatment did not improve matters. But within 36 hours of the injection of one half-billion of a mixed polyvalent staphylococcus vaccine, there was a distinct improvement in the state of the eye. Francis regarded the ocular process as embolic from the tonsils.

Francis concludes with the following words:—"Studies such as those of Reber, Brown, and Irons make it quite apparent that the former classifications of the causes of uveal disease must be re-arranged and that what has been termed rheumatic iritis is a blanket name covering a group of specific infections. Syphilis likely plays a less prominent *rôle* in disease of the uveal tract than was formerly supposed. The diagnosis of a particular case entails a consideration not only of syphilis, autointoxication, but the search for a possible focus from which infection may be tranferred to the eye by embolism,

and particularly the tonsils, alveolar processes, and accessory sinuses. The complement fixation tests for gonococcus, pneumococcus, streptococcus, staphylococcus albus and aureus, B. influenza, B. coli, and micrococcus catarrhalis are now available and should be made, and if a positive reaction is present, the appropriate vaccine should be adminstered. Post-operative or intercurrent surgical infections may offer a field for vaccine. Serologists are agreed that in every instance an autogenous vaccine is safer and more certain than a stock vaccine, but that in some infections the stock vaccines compare favourably in action with the autogenous. The adminstration of vaccine is without danger when in proper dosage and in cases when not clearly contraindicated by serious disease or kidney impairment. Vaccines should be adminstered under the guidance of one trained by experience in their use." S. S.

BOOK NOTICES.

On Blindness in Portugal. (**A Cegueira em Portugal.**) Dr. SANTOS. Coimbra, 1915.

Santos, of Lisbon, bases his pamphlet on the prevalence of blindness in Portugal on the census returns from 1864 to 1911. The first census, in 1864, made no return of the number of the blind in the country, and it was not until the second census, taken in 1878, that any definite statistics on this matter could be obtained. The third census in 1890 was the first in which a differentiation between those blind in one eye and those completely blind was attempted. The fourth census in 1900, and the fifth in 1911 have been conducted on the same lines as the third.

Statistics drawn from the second census in 1878, disclose the extremely high proportion of nearly one blind in each 500 persons in Portugal; but as indicated above, this may not mean absolute blindness, and, as a matter of fact, the returns for 1900 show a substantial reduction of these figures.

The figures in the last census show a slight increase, the numbers agreeing very closely with the results of the enquiries into the number of blind in Portugal made in 1903 and 1904 by Rodriguez and Meyer respectively. The figures for the neighbouring islands of Angora, Horta, Ponta Delgada, and Funchal, show a similar state of affairs.

A comparative table of the numbers of blind in the various European countries is given, but, since the census year differs in the various countries, it does not represent quite accurately the state of affairs at the present time. Russia heads the list with a proportion of 19·6 in 10,000 persons in the census of 1886; Portugal comes next with 13·2 per 10,000 in 1911; Spain, 11·6;

Austria, 9·09 in 1890; Norway, 8·7 in 1900; England 8·6 in 1891; Belgium, 8·1 in 1880; Italy, 7·5 in 1881; Switzerland, 7·2 in 1895; Germany, 7·12 in 1895; France, 6·2 in 1910; and Denmark, 4·4 in 1900.

Santos declares that about half the total blind in his country, are blind as the result of ocular diseases which are either curable or preventable, and ends with a weighty argument in favour of more extensive ophthalmological teaching in the Portuguese schools of medicine and a plea for governmental assistance. R. R. JAMES.

Extra-ocular Pressure, and Myopia. By ISLAY B. MUIRHEAD, M.D. London: John Bale, Sons and Danielsson, Ltd., Oxford House, 83-91, Gt. Titchfield Street, Oxford Street, W. Price 3s. 6d. net. 1916.

IT has long been believed that the effect of the action of the external muscles of the eye tends to raise the intra-ocular tension and to cause elongation of the globe. The author thinks that this doctrine is contrary to reason, and receives no support from the real facts of the case. He believes that the action of the muscles is the exact opposite, tending to cause an antero-posterior flattening.

When the recti are divided, the eyeball starts forward, showing that it is held back by the tonicity of the muscles. The impulse is not, he thinks, due entirely to the elasticity of the retro-ocular orbital contents, but partly to the elastic recoil of the eye itself. The globe being filled with fluid, and being held by some to be incompressible, it is difficult to accept this suggestion. Even if we admit a certain degree of compressibility, it certainly is not enough to act as an elastic spring forcing the eye forwards when the muscles are divided.

The second chapter deals with the evidence afforded by the change in the apparent size of objects.

According to the orthodox theory, the pressure is greatest when the eyes converge. As the eye must elongate under this pressure, the apparent size of the image should enlarge. The author states that it diminishes. We are not aware that anyone suggests that the semi-rigid sclera elongates and contracts as the eyes move. To do so sufficiently to alter the apparent size of an object, it must be highly elastic. What the theory postulates is a *gradual* stretching of the sclera by a tension which becomes somewhat raised during convergence. We fully agree with the author that there is little direct evidence in favour of this theory, and many clinical facts which render it highly improbable; but it seems equally improbable that the back of the eye is bulged inwards when the recti muscles contract, nor has the author advanced any experimental fact which supports the assumption. The change in apparent size of objects may be explained in a variety of ways, and does not afford any convincing proof of the author's theories.

In Chapter IV we are told that city dwellers are much more liable to the defect than country dwellers, the highly civilized than savages, people of sedentary lives than workmen.

In the first place—a most vital omission—the author entirely fails to separate progressive myopia with fundus changes from ordinary non-progressive myopia of low degree. We are aware that no absolute distinction is possible, for an ordinary myopia may become progressive, but the separation is, as a rule, a valid one.

All tables of statistics which we have studied, bring out one salient point: high myopia with fundus change is rather more common among the country folk who do not use their eyes than it is among town dwellers. Our experiences in Palestine have shown us that this type of myopia is quite common among the uncivilised tribes, Bedwân, Negroes, Turks, etc., who attend an eye clinic in the East. It is also seen in young children who have never read or used the needle. In fact, there seems to be little evidence that this variety of short-sight has any connection whatever with the use of the eyes. And yet the statement is constantly made that "native races are not affected with short sight." What evidence has the author for his statement that the Esquimaux do not suffer from myopia?

As regards ordinary myopia, it is natural that the short-sighted tend to adopt sedentary occupations and to gravitate to the towns; but we are not aware that there is any great difference in the incidence of myopia among the children in urban and rural areas.

The author has much to say against the practice of ordering a full correction for myopia. We gather that he thinks that persons wearing a full correction are always uncomfortable and are suffering from eye strain. Our experience is that in the majority of cases a full correction is worn with the greatest comfort. Some patients are more comfortable with a slight under correction, others again prefer to use their spectacles only when they have occasion to see clearly in the distance.

We feel that we may not have done full justice to Dr. Muirhead's book. We confess that, although we have read it twice with great care, we have not been able to follow the argument or to understand the exact object the author is trying to prove.

T. HARRISON BUTLER.

The Cantor Lectures on Optical Glass. By W. ROSENHAIN, 34 pp. Wm. Clowes & Sons, Ltd., London. 1916. Price 1s.

This pamphlet consists of three lectures delivered by Dr. Rosenhain before the Royal Society of Arts on November 29th, December 6th and 13th, 1915. They are, in our opinion, well worth reprinting, and will repay perusal.

The first lecture deals with the requirements of optical as

opposed to ordinary glass, and gives some account of the tests with which it has to comply. The remaining two deal with the progress of manufacture and the possibility of simplifying and rendering that process a more certain one.

The infinite possibilities of disaster in any one of the numerous processes at present required render the manufacture so uncertain and costly as to make the commercial side of the question of doubtful value when standing alone. The author says: "In Jena there is little doubt that financial success has been achieved by the close inter-relationship of the firm of Schott with the immensely successful enterprise of Zeiss. In addition to this profitable co-operation between the glass-maker and the optical instrument maker, the Jena firm has undertaken glass manufacture on other lines besides those of optical glass—such as laboratory ware, and more especially lamp chimneys for gas-burners. These activities, taken up by a firm originally intended to deal only with the production of optical glass, serve as a good indication of the business aspect of the manufactures in question."

After discussing the possibility of State subvention, the author comes to the conclusion that the industry of optical glass manufacture can only hold its own if put on a more rational foundation by means of laboratory researches and works experiments.

He then proceeds to discuss the more technical details of glass manufacture, and to indicate lines of research that should prove of value, notably in furnace construction and crucible material.

E. E. H.

NOTES.

Deaths.

THE untimely death of Wendell Reber, of Philadelphia, has already been announced in these columns.

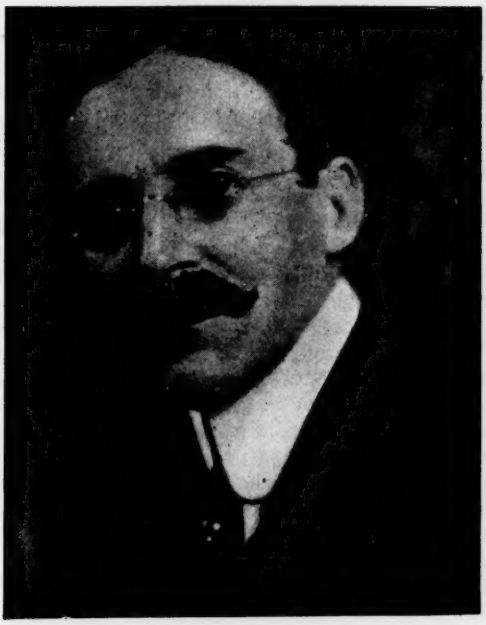
In order to show the love and esteem he enjoyed amongst his American colleagues the following quotation may be made from a letter received from Casey A. Wood, of Chicago, editor of the *Ophthalmic Record*:—

"A telegram yesterday from Wendell Reber's secretary tells me of his untimely and sudden death from pneumonia. It was a great shock to me and robbed the new year of any modicum of joy it might otherwise have held. What a charming, laughter-loving friend we have lost! I knew Wendell intimately from the earliest period of his career and loved him—as almost everybody *did* love him—like a brother, and I shall miss him more than words can express."

The following appreciation has reached us from Dr. Luther C. Peter, of Philadelphia:—

" Dr. Wendell Reber, of Philadelphia, one of America's best known and most finished ophthalmologists, succumbed to pneumonia on December 30th, 1916, at the age of 49 years.

He was born in St. Louis, Missouri, April 4th, 1867, and graduated from the Washington University with the degree of Doctor of Medicine in the year 1889. For several years he practised general medicine, and in the early 90's became resident physician in the State Hospital for the Insane, Norristown, Pennsylvania, serving that Institution for one year. The first years of



his brilliant ophthalmologic career were spent in Pottsville, Pennsylvania, whence he moved to Philadelphia and began his life-work which ended so tragically at the close of last year.

His capacity for work and his close application to his profession were in evidence from his first entrance into American ophthalmology, and he served with distinction in the Wills Hospital, Jefferson Medical College, and many other medical institutions in and about Philadelphia. At the time of his death he was Professor of Diseases of the Eye in Temple University and in the Philadelphia Polyclinic and College for Graduates in Medicine. He was Visiting Ophthalmologist to the Philadelphia, Samaritan,

and Garrettson Hospitals, and was Consulting Ophthalmologist to the Friends' Hospital and the Rush Hospital for Consumption and Allied Diseases.

Dr. Reber was a Fellow of the American College of Surgeons and of the American Academy of Ophthalmology and Oto-Laryngology, serving as President in the latter organisation several years ago. He also was Chairman of the Ophthalmologic Section of the Medical Society of the State of Pennsylvania and President of the Philadelphia Clinical Association. In 1914 Dr. Reber was the American representative of the Oxford Ophthalmological Congress and delivered an address before that distinguished body. He was a frequent and most welcome visitor at the meetings.

His contributions to medical literature were many and of great merit. The best, and the one which probably was his masterpiece, was his text-book on the ocular muscles which he wrote jointly with Dr. Howard F. Hansell, of Philadelphia, and which passed through two editions.

Dr. Reber was best known as a teacher, a special aptitude for which he possessed to a rare degree. One of his greatest interests in life was to "help and teach his boys" as he called them. His many students, who later in life became loyal friends, bear testimony to the success of his efforts in this direction.

He was a man of many sides, not only a scientist and teacher, but a man of rare culture and refinement—genial, artistic, optimistic, and enthusiastic in temperament, possessing a sense of humour which attracted to him a large circle of friends; a man of keen sense of honour and justice, and, above all, of loyalty in his friendships. American ophthalmology has been dignified and honored by his work, and his many friends have been made better by his life. Well may Dr. Reber stand on his life's record."

The following letter has been received from Dr. Darier (Paris), the editor of *La Clinique Ophtalmologique* :—

Paris, *le* 28 *janvier*, 1917.

Je suis atteré par la nouvelle de la mort de ce cher Wendell Reber. Que lui est-il donc arrivé ? Il était si plein de santé et de vie quand nous le vimes en 1914 à Oxford. Il m'avait conquis par son ardeur au travail, par sa foi en la science, et par son enthousiasme, trop rare aujourd'hui, pour nos récentes conquêtes thérapeutiques. Je le vois toujours, son ophtalmoscope électrique à la main, parcourant à Oxford les salles de démonstration, récherchant avec avidité les cas qui l'intéressaient, l'œil en éveil sous son lorgnon, le geste prompt, le verbe précis au service d'un jugement rapide et éclairé.

Combien j'aimais sa conversation pleine d'imprévu et de judicieuses appréciations !

Je le vis pour la dernière fois à ma clinique à Paris quelques jours seulement avant la déclaration de guerre, et dans sa dernière lettre que je reçus de Philadelphie il m'exprimait sa profonde sympathie pour la France à laquelle il tenait par sa mère.

L'ophtalmologie a fait en lui une perte réelle, et tous ceux qui l'ont connu conserveront longtemps son souvenir.

* * * *

The death is announced of the distinguished French *savant*, Augustin Charpentier, at the age of 64 years. He was for the greater part of his life a Professor in the Faculty of Medicine at the University of Nancy. His world-wide fame rests upon his epoch-making researches into the physiology of vision, carried out during the years 1880 to 1900, and recorded in numerous papers in the *Comptes-rendus de l'Académie des Sciences*, the *Comptes-rendus de la Société de Biologie*, the *Archives de Physiologie*, and the *Archives d'Ophtalmologie*. The results of his earlier experiments were rendered more accessible in a comprehensive treatise, entitled *La Lumière des Couleurs*, published in 1888. It is a small book, but it is rightly regarded as a classic. It records a mass of experimental work devised with great ingenuity, carried out with untiring patience and accuracy, and expounded with all the lucidity which is characteristic of the best French writings. It deals chiefly with light and colour sensations, and especially with their threshold values. In later years Charpentier turned his attention to the persistence of retinal impressions and what is now commonly known as recurrent vision. The following are a few of his observations.—He showed that dark adaptation of one eye has no effect upon the other, and that, although the fovea is night-blind relatively to the periphery of the retina, it is capable of a slight degree of dark adaptation. He was one of the first to study the photochromatic interval, or interval between the light threshold and the colour threshold for different colours. It is notoriously difficult to elicit any such interval for red, but Charpentier convinced himself that it existed under suitable conditions. He also demonstrated a photochromatic interval for colours at the fovea. Many of his experiments dealt with the relationship of the intensity of the stimulus to the area of the retina stimulated at the threshold. He confirmed and extended the observations of Ricco, and especially the law for the fovea that the total quantity of light, *i.e.*, the product of the area and the illumination, must attain a certain value in order to produce a sensation of light, and that this value is constant for a given condition of adaptation. He determined the absolute and the chromatic thresholds from the area stimulated for the fovea of the dark-adapted

eye, and showed that the law associating area stimulated and intensity is not so simple for coloured as for white light.

It is well-known that a single brief stimulus to the retina provokes pulses of light sensation. Thus, if the stimulus is given by a radial slit in a rotating disc the image is spread out into a sector which is marked with alternate bright and dark bands. The fan-like bands are known as "Charpentier's bands," and were studied by him, although, as a matter of fact, they had been previously described by Shelford Bidwell in 1894.

Enough has been said to indicate how much physiology and ophthalmology are indebted to this great French scientist, and if any ophthalmologist has not read Charpentier's fascinating little book on Light and Colours, he is strongly recommended to do so. Although published nearly thirty years ago, it is by no means out of date.

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From Paris the death is announced, at the age of 87 years, of Dr. Richard Liebreich, who, for more than fifty years, had been a naturalised Frenchman. The deceased, who practised in London for some years, and was attached (1871 to 1878) to St. Thomas's Hospital, was the author of a famous ophthalmological atlas, which ranks even to-day as a classic.

The following appreciation has been contributed by Sir Anderson Critchett:

"The recent death of Dr. Richard Liebreich at the ripe age of 87 years, leaves another gap in the ranks of the old ophthalmic Guard, and there cannot now be many remaining who were contemporary with Helmholtz, Donders, von Graefe, Arlt, Agnew, Bowman, and my father. To men of the younger generation Liebreich must be practically unknown, for, with the exception of his Atlas of Ophthalmoscopy, published in 1863, his contributions to the literature of our subject were not extensive. His excellent Atlas has been for many years an admirable medium for teaching and learning the pathological changes of the fundus oculi, and it must, in all justice, be acknowledged that during the sixties and seventies of the last century, Liebreich occupied a deservedly prominent place in the world of ophthalmology.

"After taking his medical degree, he studied for some time in the clinique of von Graefe, and ultimately became chief assistant to the great master. He then migrated to Paris, where he quickly acquired a large practice, his sudden rise to eminence being materially aided by a successful operation for cataract which he performed on the mother of the Empress Eugénie. He was at the zenith of his fame when the International Ophthalmic Congress met in Paris in 1867, and he demonstrated his then recently introduced operation for strabismus before that assembly.

The promise of a long and brilliant professional career in the French capital was unexpectedly blighted by the Franco-Prussian War of 1870. His great rival de Wecker managed to escape banishment by taking shelter behind the plea that he was of Austrian extraction, and must, therefore, not be numbered amongst the active enemies of France; but Liebreich was too recently and too intimately associated with Berlin to hope for the extension of any consideration in his favour, and he judiciously sought refuge on the more hospitable shores of England. Not long after his arrival he obtained some compensation for his previous misfortune in the offer of an appointment as ophthalmic surgeon to the recently-built St. Thomas's Hospital. In order to qualify for this it was necessary that he should hold a British surgical degree or diploma, and after an examination, in which, so rumour had it, a generous leniency was extended to the distinguished foreigner, he became a member of the Royal College of Surgeons of England. I have it on the authority of some of his former pupils that he proved to be an excellent teacher, especially on the subject of refraction, although his very limited knowledge of the English language proved a somewhat serious handicap, and in moments of excitement he often lapsed into either French or German. In spite, however, of his linguistic disabilities, he delivered an interesting lecture on "Painting," at the Royal Institution, and made two original suggestions respecting those well-known artists, Turner and Mulready, namely, that the eccentricities found in the great landscape painter's later works were due in large measure to errors of refraction which might have been corrected by suitable glasses, and that the dominant purple tone which was present in pictures painted during the last decade of Mulready's artistic career, depended on changes in the structure and colour of the lenses of his eyes. These views were much canvassed at the time in ophthalmic circles, and did not, to the best of my memory, capture universal approval.

His highly nervous artistic temperament prevented him from excelling as an operator, and in cataract extraction he adhered too rigidly to one method, for he always did a simple extraction, making his incision entirely in the cornea.

He devised a portable instrument for this operation, which consisted of a Graefe knife, a cystotome, and a small spoon, all contained in one ivory sheath, from which they could be projected as required, and although antiseptic surgery had not advanced very far in those days, he astonished and somewhat shocked my father by explaining that one of the merits of this *multum in parvo* ivory case was that it could be conveniently carried in the waistcoat pocket.

Liebreich was a past master in his knowledge of refraction, and achieved marked success in that direction, although it is worthy of note that be never employed a mydriatic.

His hobby, which he certainly rode too hard, was the frequent use of exceptionally strong prisms. At a later period, after his retirement, I had an opportunity of testing some of these cases for muscular insufficiency, and I found very few which, in my opinion, needed the amount of help which he had so lavishly given.

Although he had achieved an excellent professional and social position in London, he yearned to gain a reputation in the world of Art, for he loved painting and sculpture, and had studied the latter under the great French sculptor, Carpeaux. As soon, therefore, as he had earned a sufficient fortune he went back to Paris, now willing to acquiesce in his return, and he there passed the greater portion of his declining years as an art student, not disdaining association with the Bohemian element in the *Quartier Latin.* One object of his earnest quest was to rediscover the secret of the varnish used by the Old Masters, and I believe he was able to persuade himself that he had succeeded.

He had the gifts of gentleness and of old-world courtesy, and he has assuredly left a pleasant memory with all who knew him."

The following list contains the more important of Dr. Liebreich's literary communications:

1857. "De l'examen de l'œil au moyen de l'Ophtalmoscope." In the French edition of W. McKenzie's Treatise on Diseases of the Eye. Edited by Warlomont and Testelin.
1861. "De la rétinite leucémique et de l'embolie de l'artère centrale de la rétine." *Bruxelles.*
1861. "Scheinbare Kurzsichtigkeit bei übersichtigen Bau und Accommodations-Krampf." *Berlin.*
1862. "De la prédisposition à la rétinite pigmenteuse chez les enfants nés d'un mariage entre consanguins." *Paris.*
1863. "Atlas d'Ophtalmoscopie." *Berlin* and *Paris.* (1885: Third Edition published in London.)
1864. "De l'accommodation." Reprint from *Nat. Dict. de Méd. et Chir. pratique. Paris.*
1871. "On a new method for extraction of cataract." *St. Thomas's Hospital Reports.*
1872. "School life in its influence on sight and figure." *London.*
1872, 1875, 1878. Three lectures: "Turner and Mulready: the effects of certain faults of vision, with especial reference to their works." "The real and ideal in painting." "The deterioration of oil paintings." *London,*

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American Journals.

IN reference to the possibility of obtaining a really representative journal of ophthalmology in America, a recent issue of *Ophthalmic Literature* enquires pertinently whether the American ophthalmologists are willing to pay the price for such a publication. Our contemporary sets forth the cost of existing American periodicals, which ranges from 3.50 to 10 dollars *per annum.* With these it contrasts the prices of the large standard German journals, the cheapest of which (*Zeitschrift f. Augenheilkunde*) costs 7.50 dollars and the most expensive (v.

Graefe's *Archiv*), 20.25 dollars a year. It quotes our own subscription rate as 7.50 dollars. It is claimed that the cost of publication in both England and Germany is much lower than in the United States. On the other side of the account is to be set the fact that the ophthalmic surgeon receives better fees in America than anywhere else in the world.

* * * *

Uveoparotid Fever.

IN an editorial paragraph in the *Lancet* for January 6th, 1917, reference is made to uveoparotid fever as described by Heerfordt in 1909. Heerfordt's three patients each had bilateral parotid swelling and bilateral iridocyclitis, and two of them developed paralysis of the facial nerve in addition. From the long duration of the parotid swellings Heerfordt concluded that he was not dealing with cases of mumps, especially as there was no evidence of exposure to that infection. It seems, however, that Leeksma, of Heukelum, has met with a patient having the same symptoms as those described by Heerfordt, but in this case Leeksma found three cases of mumps occurring in the patient's environment. He came to the conclusion that Heerfordt's "uveoparotid fever" is nothing more or less than atypical mumps and is not a new morbid entity.

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Annales d'Oculistique.

THE *Annales d'Oculistique* (Paris) announces that as from January, 1917, its price will be raised to 25 francs per annum.

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Lt.-Col. W. A. Turner, C.B.

THE honour of Companion of the Order of the Bath has been conferred upon Temp. Lt.-Col. William Aldren Turner, once secretary of the Ophthalmological Society.

* * * *

Oxford Ophthalmological Congress.

THIS Congress will assemble at Keble College, Oxford, on the evening of Wednesday, July 11th next, and the meeting will be held on Thursday, the 12th, and Friday, the 13th July.

The first day, Thursday, the 12th, will be devoted to a discussion on "The Correction of Errors of Refraction," to be opened by Dr. George M. Gould, of Atlantic City, N.J., U.S.A., and by Lieut.-Col. R. H. Elliot, of London.

The official dinner of the Congress will be held in the hall of Keble College, on the 12th July, when the Very Reverend T. B. Strong, Vice-Chancellor of the University, will preside. The Doyne

Memorial Medal will be presented to Dr. George M. Gould on that occasion.

* * * *

The Lucien Howe Prize.

THE Medical Society of the State of New York announces the following subjects as suggested for the Lucien Howe Prize of one hundred dollars, although an author may choose his own subject if he so desire :—

THE LUCIEN HOWE PRIZE.

Description of diseases of the eye (especially optic nerve lesions) found in syphilis of the nervous system, stating the forms occurring in cerebro-spinal syphilis and para-syphilis.

How far should the State go in providing examinations and treatment of eye diseases seen in children in public, parochial, and private schools ?

Experimental or clinical study of the extent to which the vitreous may be manipulated or transplanted.

Essays must be in the hands of the Committee not later than March 24th, 1917, and should be sent to the Chairman, Albert Vander Veer, 28 Eagle Street, Albany.

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Appointment.

MR. JOHN R. ROLSTON has been appointed honorary consulting ophthalmic surgeon to the Plymouth Garrison, Southern Command.

FUTURE ARRANGEMENTS.

	1917.	
Egyptian Ophthalmological Society	March 2nd, 9 a.m.	Medical School, Cairo.
Midland Ophthalmological Society	April 3rd.	Birmingham.
Ophthalmological Society of the United Kingdom	May 3rd, 4th, and 5th.	London.
Midland Ophthalmological Society	June 5th.	Oxford.
Section of Ophthalmology, Royal Society of Medicine.	June 13th.	1, Wimpole Street, London, W.
Oxford Ophthalmological Congress	July 12th and 13th.	Keble College, Oxford.